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ESCAPE FROM THE PAST.[1]

By THE HONOURABLE SIR GARFIELD BARWICK, Q.C., M.P.,
Attorney-General of the Commonwealth of Australia.

I FEEL most honoured this evening in being asked to deliver this the eighth Maitland Oration at the invitation of the Sydney Hospitallers. Those who have preceded me in this great privilege have without exception been most distinguished in the art and practice of medicine, and have been able, by their orations, to make substantial contributions to the total knowledge of medicine, medical education or medical techniques. When I list my predecessors, as I shall shortly do, you will appreciate my trepidation in thus standing, even for so short a space as I shall stand, here in their footprints, if not, in their shoes. This institution—for such the Maitland Oration has certainly become—owes its origin to the decision of the Old Sydney Hospitallers' Club, as the Sydney Hospitallers were then known, a decision taken by the Club in 1935 when under the presidency of Dr. H. K. Porter to commemorate the work of the late Sir Herbert Lethington Maitland, and, in particular, his work in connexion with the Sydney Hospital. After its interruption by war, the oration has been delivered in each of the last six successive years as well as in 1935. Sir Robert Hutchison, Sir Raphael Cilento, Sir Heneage Ogilvie, Dr. Donald Hunter. Professor Charles H. Best, Sir James Learmonth and Professor Keith Inglis have in turn, and in that order, graced the rostrum and made their significant contributions.

My training is that of the lawyer, though perhaps my interests have ranged more widely than the confines of Blackstone's "Commentaries" or Halsbury's "Laws of England". Of medicine, I know so much as the cross-examination of physicians and surgeons, when giving evidence both descriptive and opinion, has required—knowledge special in its nature and extent, bent to serve a point of view and both superficial and ephemeral. Of clinical medicine, I know no more than observation and curious interrogation as a patient has allowed. This is the jubilee year of the clinical school of Sydney Hospital. To that school Herbert Lethington Maitland was appointed first lecturer in clinical surgery. It therefore seemed to me that I should devote myself this evening to some of the historical facts associated with the notable, if not bizarre, beginnings of the building, which saw the early struggles of the hospital and at least part of its rise from the simplicity, if not the crudity, of the early 1800's to a much more complicated and sophisticated state in the late 1800's, when it began to be replaced by that imposing pile of masonry which now houses it. Consideration of these few facts of our history will lead me to make some observations on the effect which the origins of the hospital and, indeed, of our early community, have had on the attitude of our people to the provision of money for the construction and maintenance of hospitals and to the cost of hospitalization. In this wise I may unite some facets of the early days of hospital development and of the

[1] The eighth Maitland Oration of the Sydney Hospitallers, delivered on October 23, 1959, in the Great Hall of the University of Sydney.

early days of the administration of the law, thus finding some field for a contribution to this gathering and bridging to some extent the abyss which separates me from the distinction of my predecessors in the delivery of this oration.

As I have said, this oration was instituted by the Sydney Hospitallers, now a group of graduates and medical officers associated with the Sydney Hospital. This Club (for it is an unincorporated association of individuals) began as the Old Sydney Hospitallers' Club in July, 1928. Intended to be composed of residents of the Sydney Hospital and to fulfil the functions of a social club, it has developed into a significant force in the medical education of this State, giving encouragement to students both at the undergraduate and the graduate level. In 1958 the basis for membership was extended so as to embrace all graduates of the Sydney Hospital Clinical School—the great instrument of medical teaching which was formerly inaugurated and recognized on October 20, 1909. At the same time in 1958 the Club's name was changed to that which it now bears, "The Sydney Hospitallers".

How wise was the decision of the Hospitallers to commemorate Herbert Lethington Maitland by an annual oration! For whether it be good or ill, outstanding or indifferent, the delivery of the oration is by a living being. Of necessity, it is heard and received by living creatures who have assembled as people with affection and with respect for the occasion, have assembled as much to remember and to commemorate the name of Herbert Lethington Maitland as to hear what the orator has to say. Truly a living and self-renewing, satisfying memorial! How appropriate such a memorial for this man of vitality, this man of action and of practical human consequence!

Herbert Lethington Maitland was a native of this State, born at Tumut in 1865. He came to the University of Sydney in 1887, at the age of nineteen. He did not appear on the academic scene as a pale and sickly scholar, not "sicklied o'er with the pale cast of thought". He came from Newington College with a school-boy record of full-blooded participation in sport, with the acceptance and acclaim of his fellows as an all-rounder, as a personality.

He moved through a medical course balancing an excellent record for knowledge and scholarly achievement with days of full participation in the sporting and social life of a university, then, because of its small numbers, in its most intimate and, perhaps for the student, its most satisfying era. He graduated in 1892 with second place in surgery. Although not the first graduate of the Medical School of the University of Sydney to become a resident medical officer of Sydney Hospital—a distinction given to A. G. Henry, M.B., Ch.M., who was appointed a resident in the hospital in the year 1888—he was the first graduate of the Medical School of the University of Sydney to be senior resident and deputy to the superintendent at the hospital, a position to which he was appointed in 1893. He practised privately in Elizabeth Street, Sydney, from 1894, and later in Macquarie Street. He acted both as house surgeon and as house physician to the hospital for a considerable time and was appointed an honorary surgeon to the hospital in 1902.

When the clinical school was founded at the hospital in October, 1909, Maitland became its first and one of its most distinguished clinical lecturers in surgery. And so he remained till 1923.

His biography I know is writ large in your minds, for you gather here to revere his name and by your presence to pay your tribute to his distinction and to his attainments. As I read his biographical references, there emerges a personality, direct, competent, practical — a social being attuned to his fellow-men, pleasant and acceptable in his dealings with them, capable of assisting them, but perhaps above all capable of leading them. There emerges also that truly rare human, a great teacher: Herbert Maitland leaps from the page of history, not only as one who has informed himself, who in his lonely vigil has pushed back for himself the barriers to knowledge, one who has developed great dexterity of hand and unique coordination of mind and muscle, one with a high degree of sensitive reaction, with that executive and moral courage and resource which go to make the great surgeon, and as one with consummate powers of observations and a retentive memory; for all these he was and had, but he emerges as one who has so mastered that which he has learned, who has found such confidence in his own techniques and methods, that he can with directness, with simplicity and with contagious confidence communicate to others all that he knows, all that has become a part of himself. That he should have had the will, inclination and ability to do so make him for me an outstanding man; for in my philosophy, the giants of mankind are not those who simply discover, but those who, having discovered, both desire to tell and have the ability to tell their fellows in simple and compelling terms the reality of their discovery. Thus do we advance as humans in the unending process of widening the horizons of understanding and of comprehension. And so it was with Herbert Maitland. He was a great teacher. He taught in the clinical school of Sydney Hospital, whose alumni are found in the Sydney Hospitallers.

Herbert Lethington Maitland was knighted in 1915 for his services to the community, chiefly, though not exclusively, in the practice and the teaching of medicine. He worked hard in his private practice, passing progressively from general practice to operative surgery as his speciality. He was unceasing in his efforts to improve the surgical wards of the hospital. He found time for his professional association, being for many years a member of the Council of the New South Wales Branch of the British Medical Association and its president in 1911 and 1912. Yet, withal, he was with us but 55 short years; of working life, from graduation in 1892 till his death in 1923, he graced the scene but 31 years; yet he made an indelible mark. Though constantly active, he is said rarely to have appeared tired; but his expenditure of himself, without reservation or calculation of the daily toll on his physique of the energetic routine which he set for himself, assisted no doubt by the after-effects of pneumonic influenza of 1919, caught up with him on July 23, 1923, when he died suddenly, in his professional rooms in Macquarie Street—so suddenly, indeed, that no time was afforded to obtain medical assistance for him. To one who evidently enjoyed labour, if end there must be, such an end in the midst of his daily work would not have been a source of complaint. In his obituary in THE MEDICAL JOURNAL OF AUSTRALIA I read an eloquent and revealing sentence, a tribute to essential greatness and to a fullness of life and of achievement; his biographer with classic simplicity says: "He knew his work well and was not afraid of hard work."

This year—indeed, almost to this day—marks the fiftieth anniversary of the establishment of the clinical school of the Sydney Hospital, the school which owes so much to him as its first clinical lecturer in surgery. Others, much better qualified than I, have emphasized the basic importance of such a school in a hospital as well provided with clinical material as the Sydney Hospital has been and still is. But let me say that I feel that nothing, no tutorial substitute, can take its place in the training of the medical man. Through that school have passed and are still passing men of oustanding capacity, receiving from it indispensable training for their lifetime of effective and conscientious service to their fellow-men in their times of injury or ill health. In his lifetime Sir Herbert Maitland was an inspiration to them, and his memory remains an abiding challenge.

It is fitting that I turn now for a short while to the origins of this hospital, in which Sir Herbert Maitland spent so much of his working life, and with which the clinical school is associated.

On December 28, 1809, Lachlan Macquarie arrived in Sydney Cove as the Governor of the Colony of New South Wales. He was not intended or trained for such a responsibility, and it fell to him by the chance that Brigadier-General Miles Nightingall, who was to have been the Governor, was unwilling to proceed here to take up the office. Lachlan Macquarie was Commandant of the 73rd Regiment, which the British Government had decided to

send out with the intended Governor, Miles Nightingall, to replace the New South Wales Corps, the 102nd Regiment, which had been recalled because of the part which it had played in the rebellion against Admiral Bligh. Macquarie did not look forward to his assignment as the Commandant of the regiment then posted to the penal Colony of New South Wales. He felt that he was to suffer something akin to banishment or transportation itself. But when Nightingall, whose right arm and wrist, affected by rheumatism, seemed to incapacitate him from writing, declined the appointment as Governor, Macquarie was to sail to the Colony as Lieutenant-Governor to await the arrival of a replacement for the unwilling Nightingall. However, Macquarie pressed Castlereagh for the appointment of himself as Governor. Unexpectedly, at least as far as his own real anticipations went, he was successful. He was appointed to this office and sailed as the Governor-elect of the Colony. Castlereagh, who appointed him, when speaking later in Parliament in defence of Macquarie, said that the only recommendation which he (Macquarie) had possessed (at the time of his appointment as Governor) had been his merit. From all that had since been heard of him, it seemed that he had completely fulfilled the sanguine expectations entertained of him. As one looks back over the scene, it can be said that perhaps a little tempering of political experience and of the gentle and indispensable art of compromise might have carried Macquarie much further than the peaks to which in fact he attained, and would perhaps have given this country an even more liberal impetus than it received from his all too short regime.

Macquarie found the Colony in disarray. He reduced it to order and set it on the road to achievement. But it is not my purpose to deal with his virtues in general, or with the general social conditions as Macquarie found them or as he left them. Suffice it to say he did well, and to him we owe much.

In the years which had followed Phillip's establishment of the settlement in 1788, the valley of the Tank Stream had been to a large extent cleared, and there were dotted on the hillside little houses with their walled enclosures. Malcolm Ellis, in his notable biography of Lachlan Macquarie, thus describes the scene at the time of Macquarie's arrival:

> And rude and irregular though Sydney township itself might be, an irregular village of houses, cottages and bark huts with perfectly naked savages peeping through unguarded windows at the very gates of Government House itself, it had a certain vivid liveliness when one viewed it from the peninsula to the east of the Cove where endless generations of aboriginals had left great mounds of half buried sea shells from their feasts . . . . Every house had its trees and its garden and to the diversity of whitewashed stone, yellow brick and wattle and daub was added the brilliance of high-running ensigns above the fort and public buildings and sound and movement from the swinging sails of windmills etched against the skylines above the ridges . . . . There were no streets in the true sense of the word and the general effect, though pleasantly informal, was not orderly. There was . . . a rotting hospital whose walls had been put together in England and carried 12,000 miles across the ocean.

Macquarie, in his report to Bathurst in July, 1822, wrote:

> I found the Colony barely emerging from an infantile imbecility and suffering from various privations and disabilities, the country impenetrable, agriculture in a yet languishing state; commerce in its early dawn; public buildings in a state of dilapidation and mouldering to decay; the few roads and bridges formerly constructed almost impassable; the population in general depressed by poverty; no public credit, no private confidence; the morals of the great mass of the people in the lowest state of debasement and religious worship almost totally neglected . . . .

Macquarie thus found the provision for hospitalization grossly inadequate; or, perhaps, even so strong a statement is itself too great an understatement. The premises at Dawes Point, of which I have just quoted Malcolm Ellis's description, were lamentably too small. Patients were housed in tents about the hospital premises, necessary drugs were in such short supply that local plants and herbs were called in as substitutes. But, and perhaps from the point of view of something I wish to say, most significantly, the official attitude, ultimately personified in Bligh, towards hospitalization and the treatment of the sick bordered on the barbarous. The hospital and the medical staff were provided for those for whom the Government was responsible, and it was "always a moot point whether it was the duty of the medical staff to attend either midwifery, settlers or other free men who were not victualled by the Crown".

It must be remembered—indeed, I suppose Sydney-siders are not often allowed to forget—that the Colony commenced as a convict settlement. It was only by almost imperceptible steps that any other purpose for government expenditure obtained justification. Phillip may have had visions of greatness for the country as a place in which free men could dwell, and by their achievement and enterprise add lustre to the name and fame of England and increase her natural wealth and international prestige; Macquarie saw the vision splendid with greater practicality and consequence.

But the heart of the Empire, and the source of the necessary funds to begin the long trek to economic sufficiency and national wealth, had no such aspirations—no intention to do more than service, at a meagre and indeed parsimonious level, a penal settlement, a place to hold those whom the law of the day had found to be socially undesirable. This I say in no carping or critical vein, for I understand, and indeed smpathize with, those who, sitting in London (infinitely much further away from this place then than it is now), depending only on conflicting dispatches and correspondence, much of it written more in point of advocacy or prejudice than in description of objective fact, saw no reason to squander the British revenues on what looked so like a pipe dream. The marvel is not that so little was done, but rather that in the fullness of time so much was done to advance this land beyond the stage of a penal settlement. But for what I have to say, it is important to remember the effect on the early institutions, and particularly on the General Hospital, which this understandable attitude of mind, both in the Home Government and in the local population, necessarily had.

So apparent was the need for a hosiptal, and so executive was Macquarie, that within eight weeks of his arrival, in a dispatch dated March 8, 1810, which he sent to Viscount Castlereagh, he wrote:

> There will be an absolute necessity for building a new general hospital as soon as possible, the present one being in a most ruinous state, and very unfit for the reception of the sick that must necessarily be sent to it, of which there are on an average seldom less in it than between seventy and eighty men, women and children.

By May 17 of that same year, 1810—that is, within three months of the date of the dispatch to Castlereagh and within four and a half months of Macquarie's arrival in the Colony—a public notice appeared in the *Sydney Gazette* over the signature of D'Arcy Wentworth, who had been appointed Acting Principal Surgeon, in the absence of Principal Surgeon Jamieson in England to attend Major Johnson's court martial. The notice foretold the calling of tenders for the erection of a General Hospital, with medical officers' quarters. It indicated that plans could be inspected at D'Arcy Wentworth's house within 14 days. Its precise terms are worth recalling.

> Government Advertisement.
>
> It being His Excellency the Governor's intention to Contract for the building of a General Hospital, Medical Officer's Quarters, with the necessary Offices and Inclosures,—Notice is hereby given that a Plan of the above will be ready at my House for the Inspection of those desirous of contracting for the same, in Fourteen days from the date hereof. By Command of His Excellency the Governor,
>
> D. Wentworth,
> May 17th, 1810. Acting Principal Surgeon.

Whether the plans were ready within that 14 days or not does not appear, but the plans themselves are extant, both the ground plan and the elevation. They depict three noble buildings of ample and pleasing proportions. The authorship of the plans is obscure, there being some inclination to credit Mrs. Macquarie with their devising. As I have said, three principal buildings, each of two stories, were provided, a central building containing eight wards and two flanking but independent buildings to house the medical staff. In the main building there were four wards each 58 ft. 6 in. by 24 ft. in area, and four wards each 61 ft. 6 in. by 24 ft. in area, the ceilings in each case being 16 ft. high. The intention was to accommodate 20 patients in each ward, thus providing approximately 1150 cu. ft. per occupant. The plans were executed substantially as drawn. The two flanking buildings in broad outline remain with us. The northern building, after a great deal of addition, now houses the Legislative Assembly, and the southern building almost in its original state, the Land Tax Department. The foundation stone of the main building was laid by Macquarie on October 30, 1811, and as far as is known, the hospital opened for business in the middle of 1816.

When completed, the buildings were not wholly devoted to hospital purposes, and the wards were not limited to 20 patients. Dr. Watson, in his "History of the Sydney Hospital", states that the hospital was constantly overcrowded, and that wards intended for 20 held as many as 42 at one time, with beds arranged down the centre as well as along the walls of the ward. He writes:

> The general condition of the hospital was disgraceful. After the completion of the building it was found that a mortuary had been forgotten, and to supply this want one of the kitchens was converted into a dead house. As the other kitchen was occupied by the overseer and attendant there was no available place for the hospital cooking. All food was in consequence cooked by the patients themselves actually in the wards, and as rations were issued to every patient only three times a week each ward was virtually a scullery, kitchen and larder combined in addition to accommodating patients.

As, of course, most of the patients were prisoners, the hospital buildings were surrounded by a wall 8 ft. high, and as Dr. Watson writes:

> The patients were mustered every evening by the overseer who locked them in the wards at the hospital at sundown. They there remained until the following morning at 6, and so were shut without overseer, nurses or attendants throughout the night, and the abuse that accrued under such conditions may be readily conjectured when we remember the depraved class from which the patients were drawn . . . .
>
> No lavatories had been provided in the hospital itself. The patients when too weak to walk were seen crawling on their hands and knees to those outside. Patients were often allowed to lie in bed without clothing. Dressings were thrown under the bed and left there. All linen was changed once a week and replaced by imperfectly washed linen. Convalescents were allowed to do their washing in the wards . . . . and until the Governor noticed the practice and prohibited it . . . . were allowed to hang their linen to dry on the hospital verandah.

The hospital had been grandly conceived, and if wholly utilized for hospital purposes, might have been adequate for the needs of the growing community. But, in fact, the hospitalization, if I may grace the treatment which I have described by so noble a word, was almost as deplorable as the conditions which Macquarie found in and around the original hospital at Dawes Point on his arrival.

Two features of the building and its site have been an embarrassment to the hospital and to its hospitallers throughout the later years of the hospital. Macquarie chose the ridge on the eastern side of the valley of the Tank Stream, to the south of the Government Domain, as the place on which to construct the new General Hospital. It was a lovely site then—and it still is. He showed excellent judgement in moving the hospital from the site on the lower ground near the water at Dawes Point to this high ground close to areas intended to be kept permanently as open areas—the Government Domain to the east and to the north, and Hyde Park to the south. The main building was designed to be on the very crest of the ridge, a little behind the alignment of the flanking buildings on the crest of the ridge. Macquarie opened a new street—the first straight street in Sydney—to service the hospital. He called it Macquarie Street. None were then to know that the most important street of this great city was to develop at right angles to the new street, Macquarie Street, and ultimately to eat its way eastwards to a junction with it, directly opposite the site of the central building of the new General Hospital. The excellence of the site in itself, as it gazes down the line of the valley of the Tank Stream, became a provocation to the town planners. The retention of the Government Domain at the rear of this site has done little to divert the planner's interest.

Dr. Fiaschi, in his inaugural address delivered at the official opening of the Sydney Hospital Clinical School for Medical Students on October 20, 1909, recounts:

> The '80s was a very critical period for our history. Its very existence was at one time in question. Proposals were freely discussed in the newspapers that the valuable land on which the old hospital was standing should be resumed by the Government and put to other public use. I remember having been present at a meeting of medical men at which an objection was sent to the Government of the day requesting that the Sydney Hospital should be left on its old beautiful site.

But one other circumstance aggravated the embarrassment of those who, like the Sydney Hospitallers, have a special, if not a sentimental, interest in the maintenance of the Sydney Hospital on that site.

Only 40 patients were accommodated in the hospital at the outset. Only three of the eight wards which were provided in the main building were used for housing patients—three on the southern end, two on the ground floor and one on the first floor. The fourth, on the southern end of the building, was used as a dispensary for hospital purposes and as a store for hospital supplies.

The main building was so constructed that it had two separate staircases, one serving either end. It could thus be easily used for two distinct purposes, entrance being gained to either end by a separate doorway. The claims of the law for accommodation intruded from the very beginning. In the result, the law practised in one end of the building and medicine in the other. Thus I have some claim to an interest with the Hospitallers in the origin of this hospital.

A Supreme Court of New South Wales was first set up by what lawyers call the Second Charter of Justice—that is to say, by Letters Patent bearing date July 4, 1814. These Letters Patent provided for a Supreme Court and for a Governor's Court. A judge and two magistrates nominated by the Governor were to constitute the Supreme Court, and a Deputy Judge Advocate-General and two local residents to be nominated by the Governor were to constitute the Governor's court. Previously there had been only military courts. These continued for criminal purposes. Jeffery Hart Bent had been appointed the Supreme Court Judge, and his brother, Ellis Bent, the Deputy Advocate-General.

But in truth, the question of the use of the hospital for Courts did not remain a continuing question. A quarrel developed between Jeffery Bent and the two magistrates who had been appointed by Macquarie to sit with him, Broughton and Riley, which completely stultified the work of the Supreme Court. The magistrates, no doubt supported by Macquarie, desired to admit as advocates before the Supreme Court two ex-convicts, by name Crossley and Eagar. Jeffery Bent refused to accept these ex-convicts in the Court and refused to sit. Consequently, until the arrival of a new judge in 1817, the Supreme Court did not function at all under the Second Charter of Justice.

The northern end of the new main building of the hospital was offered to Jeffery Bent and his brother for the purposes of a Supreme Court and the Governor's Court; but neither Jeffery nor Ellis Bent would accept it. Apparently Lord Bathurst had discussed with Jeffery Bent,

before he set out for the Colony, the possibility of using the northern end of the hospital building for the purpose of the administration of justice; but when here the Judge and the Judge Advocate regarded the offer as of nothing better than "a common hospital ward", which they claimed to be in a most disgraceful state from dirt and vermin. In short, they spurned the suggestion that the building was in any part fit for the administration of the law by them. It is amusing to recall that the *Sydney Gazette* of the day, in relation to the attitude of the Bents, acclaimed the proportions of the building and referred to the verandas supported by pillars of good dimension.

It would appear that at least from the middle of 1816 till well into 1817 the northern end of the main hospital building was unused, notwithstanding the conditions at the southern end which I have described.

In the end, Jeffery Bent was recalled. In 1816 John Wylde arrived as the new Deputy Advocate-General, and Barron Field arrived in February, 1817, as the new Supreme Court Judge. The four wards in the northern section of the building were then given over to the housing of the courts, the two lower wards being used as court rooms with necessary ante-rooms, and the upper wards being intended for the accommodation of the judge. In fact, the judge was content to use one ward as his chambers, and oddly enough permitted an artist to use the other, or at any rate portion of it, as a studio. Amongst the earliest cases to be heard in the civil jurisdiction set up under the Second Charter of Justice was the famous case brought by the Reverend Samuel Marsden for libel against Campbell, the Governor's secretary, an action heard by Mr. Justice Field, in which he gave a verdict for Marsden for £2000 by way of damages. Mr. Justice Field no doubt sat in the hospital building to hear this *cause célèbre*. Prior to this civil case of libel, there had been a proceeding for criminal libel against Campbell before the new Deputy Judge-Advocate-General and three red-coated officers of the then newly arrived 48th Regiment. This had ended inconclusively. But the main building of the hospital was not used for this proceeding. Wylde sat in portion of the northern building, which now houses the Legislative Assembly.

The Supreme Court thus remained in occupation of the northern half of the main building of the hospital until May, 1824. The Statute of Geo. 14 C.96 of 1823 both set up and provided for the granting of a new Charter of Justice. That Charter, which is sometimes referred to as the Third Charter, and is that under which the Supreme Court of New South Wales still functions, was issued on October 13, 1823, but, of course, it did not arrive in the Colony for a considerable period thereafter. In fact, it arrived on March 5, 1824, but was not officially published until May 17, 1824, when it was read at the opening of the new Supreme Court in the Georgian School, which then and for many years later was entered from Castlereagh Street. The Supreme Court still sits there, though nowadays there is no entrance from Castlereagh Street to the original portions of the building, the principal entrance being in King Street through the arcading which was subsequently applied to the original school building.

But the removal of the Supreme Court from the northern wards of the general hospital—or as it became, the Sydney Infirmary and Dispensary—*did not remove the embarrassment* which the law has had for Sydney Hospitallers. From time to time the use of the site, always a commanding one, and much the more commanding these days as it looks down Martin Place, has been canvassed by the lawyers for the purpose of erecting an imposing Court House to house the Supreme Court of New South Wales. As a lawyer interested in the majesty of the law which a great building can embody, I, of course, have some sympathy with these recurrent claims. Indeed, as Hospitallers will remember, the claims of the law had been pressed to the point of the preparation of plans for that use of the site, plans which are still in existence. Hence what I have referred to as Macquarie's excellent judgement in the choice of the site. Bathurst's ideas of using portion of the building for the administration of the law have, from time to time, brought anxiety and disquiet to the medical profession and particularly to Sydney Hospitallers.

At the outset the two flanking buildings were used to house the surgeons and a dispensary; but in 1828 the principal surgeon finally vacated the northern building, and the Legislative Council, which had been set up by the Statue of 1823, laid claim to its use and entered into occupation of the northern building in 1829. The Legislative Council and its successor, the Legislative Assembly of New South Wales, have since that time had the use of that portion of the original hospital buildings.

The southern building was not conveyed to the present hospital when the hospital was incorporated by the *Sydney Hospital Act*, 1881, and came to be used for governmental purposes, successively as a mint, to house the officers of the Housing Commission, and latterly to house officers of the State Land Tax Department.

Time does not permit reference to the activities of the years in which the hospital outgrew and progressively supplemented and ultimately replaced the original main building of which it had the full use from 1824 onwards. Suffice it to say that by 1907, with the opening of the Renwick Pavilion, the reconstruction of the old hospital was complete, and all substantial trace of the original main building was gone.

Later Macquarie Street was widened, and some of the open ground in front of the hospital was lost to it.

Let me now turn from thus reminding you of some of the more interesting facts about the use of the original buildings of the General Hospital of 1816, to recall to you the basis of early hospitalization and the method of financing the hospital, both in point of construction and in maintenance. I do this because I feel that this early basis has had, and still continues to have, a marked effect on the attitude of our people to the provision of hospitalization.

Sydney Hospital was built to afford hospital facilities for convicts (including those who had been assigned to settlers), for soldiers, to some extent for seamen in service, and for public servants, for all of whom, in one capacity or another, the Crown was responsible. In short, the hospital was to provide hospitalization for all those who were or could be placed on government stores. All these groups felt—and rightly felt—that they had a claim on the "Government" for their hospitalization. The "Government" to which they looked was in a real sense external to the community of which they formed part. Their reliance on the Government in this respect was no invasion of their independence. Indeed, most of them had little independence worth speaking of. In their hospitalization wholly at government expense, no element of charity obtruded itself. They were no more than receiving their due.

Over the succeeding years, not merely self-government, but complete self-dependence in this community was achieved. The habit of reliance on the "Government" derived, as I feel, in no small part from these origins has persisted. The change in the nature of the "Government" on which reliance is placed has not been sufficiently perceived. And now that the "Government" is no longer external to the community, but is representative of the community itself, the revenues available to and expended by the Government are no longer those of a taxpayer of another community as in those early days, but are revenues derived from the taxpayers of this community itself. Their collection and expenditure represent a distribution of financial burdens over the individuals and groups of the community. That we have formed a federation has done nothing to eliminate the misconceptions spilling over from the past. The Federal Government is regarded as external to the State, and vice versa. The federal concept is at best difficult enough; but the pervading notions drawn from our past make its comprehension even more difficult. I shall later have just a word to say on the significance of this change in relation to hospitalization.

First let me remark on the initial method of financing the Sydney Hospital, both in point of construction and in

point of maintenance. The terms of the building contract under which the new General Hospital was constructed are very striking. Macquarie's own resources as Governor of the Colony clearly could not provide a cash payment for the building cost of so imposing a structure as the plans for the new General Hospital represented. Though the use of hard spirits as currency was to be stamped out, Macquarie financed the construction of the building by the grant to the builders of an exclusive licence to import a stated quantity of spirits, 60,000 gallons in all, together with the use of a certain number of convicts and a certain number of draught cattle and a stated number of oxen for slaughter for rations. Hence, the almost affectionate appellation of "rum hospital" which the original Sydney Hospital bore.

It is, perhaps, a tribute to the skill of the lawyer in preventing the disclosure of inconvenient facts that whilst the building thus financed at the one time housed the courts and the hospital, I have never heard the expression "rum courts" or "rum justice". I have heard only of the "rum hospital".

The running expenses of the hospital were met out of colonial revenues in the ordinary course. Some small expenses were in fact met out of a fund raised by local taxes and in the nature of a local government charitable fund. The salaries of the surgeons were met by the Government, though the surgeons themselves were allowed a degree of private practice. As I have mentioned, anybody who was "on" the Government stores, being victualled by the Government, could be admitted to the hospital and treated without payment of any kind. Convicts who had been allotted to a settler might be admitted on terms of the settler finding their ration for 14 days. At the end of 14 days the settler could return the convict to the Government—that is to say, put him back "on" the Government stores—and thus at the one time relieve himself of any further liability and put the convict patient in a position to claim his further hospitalization gratuitously and as of right from the Government.

In the intervening years, Sydney Hospital has had to depend to no small extent on benevolence for its maintenance and upon the devotion of many right-minded citizens for the support and management of the hospital. This has enabled many patients to receive hospital treatment, either gratuitously, or at any rate for a charge which was less than the hospital's cost of providing it. Latterly we have had more government assistance and the hospital benefits scheme with the participation of the various funds, which have undoubtedly greatly assisted in the situation.

One cannot help wondering why, in a community as robust and individualistic as this one undoubtedly is, our people do not regard the expense of hospitalization as being something which they should provide for themselves, whether by saving or by insurance. The sudden and unexpected nature of illness and the need for hospitalization do not excuse but rather demand that provision be made.

I think most would agree that it is right and proper that the whole community should contribute to the construction and equipment of hospitals, to the maintenance of their fabric and to the replacement and modernization of the equipment. That this burden should be cast on the revenue is understandable, though, of course, it still does the community honour and the individual credit that those who have sufficient wealth to enable them to do so should make benefactions for hospital structure and equipment.

But why is it that our people call on the "Government" to provide hospitalization without payment of what is the true cost of running a hospital? I suppose our people dislike the idea of charity more than most. We glory in and boast of our pioneering strength, of our personal achievement; the self-made man, who by his own efforts has bettered his lot, has increased his knowledge and capacity and his station in life and has gathered some material wealth, wears the aspect of a hero with us. Why, then, should the care of one's own person in ill health be thought to be to any extent the obligation of the next fellow? And why is not a charitable quality, present in a payment by the revenue of the deficiency in the running cost of a hospital, perceived and resented by so proud a people as we undoubtedly are? In countries where abject poverty is rife, where no middle group exists between the extremely wealthy and the abject poor, one can readily understand the willingness of the poor to accept the benevolence of a government which draws its funds from the wealthy minority. But in this land, is it not true to say there are no necessary poor? The great bulk of the population is of the middle group. Extreme wealth is unusual. Why, then, do our people overlook the need to provide against the almost certainty of hospitalization for themselves and their family? If the maintenance of the buildings, the amortization and replacement plant were treated as outside and not part of the running costs of a hospital, the commercial accounts of hospitals—excluding teaching hospitals with their peculiar problems—should show figures of cost which the average man in receipt of wages or salary should be able to provide against from his own resources by way of regular saving or participation at an adequate level of subscription in insurance or benefit schemes. The service which the medical profession gives to our public hospitals in an honorary capacity makes this result much the more possible; but our people still call on "the Government" as if it, the Government, stood in the same relation to them as did "the Government" for whom the Sydney Hospital was intended at its inception to provide service in time of illness. I feel that this attitude of mind, this failure to assert a sense of independence and personal pride, is a very real result of our origins as a penal colony, but particularly the result of the circumstances to which I have called attention—the purposes for which and persons for whom the General Hospital originally was built in 1816—and of the sources from which its running expenses were drawn. There lingers still in our minds a conception of government which is external to our community, and which, being external, owes us duties, and from which we derive rights, that relieve us of being in these necessary respects self-reliant.

My paper has wandered from the occasion of this day of jubilee celebration of the inauguration of the Clinical School of Medicine at the Sydney Hospital in association with the University of Sydney and from the commemorative purpose the paper was intended to serve. I return in conclusion to the occasion and to the commemoration. Herbert Lethington Maitland typified the service rendered without stint by the medical graduates of our University to a community which, as I have mentioned too often, seeks their gratuitous services as of right and too easily denies itself the satisfaction of paying its own way. So hard it is to escape from the past.

INVESTIGATION OF BLOOD DONORS IN ACCIDENTAL TRANSFUSION MALARIA — PLASMODIUM VIVAX, FALCIPARUM AND MALARIÆ INFECTIONS.

By ROBERT H. BLACK, M.D., D.T.M. & H., M.R.A.C.P.,
School of Public Health and Tropical Medicine, Sydney.

DESPITE measures taken to prevent accidental transfusion malaria, such cases occur from time to time. This paper describes the investigation of blood donors and the identification of those who had made donations of blood containing malaria parasites in a series of cases of accidental transfusion malaria in Sydney in recent years. The inquiries were made at the request of the Director of the Red Cross Blood Transfusion Service, Sydney. The adjective "accidental" is used to distinguish the condition from deliberate transfusion malaria as in malaria therapy and experimental subinoculation malaria.

In all the cases examined the diagnosis of malaria had been made when the writer was consulted, and in all but one the origin of the infection in previous blood transfusions was already suspected. In the *Plasmodium malariæ* infection this was not so; the possibility of this type of origin was suspected during consultation with the attending physician when neuritic complications were being considered.

The procedure followed was the same in all cases: the numbers of all the bottles of blood used for transfusion, which were recorded on the case histories of the patients, were referred to the Transfusion Service, and the names and addresses of the donors were obtained. All the donors were visited (with one exception) and questioned concerning possible attacks of malaria or residence in a malarious country. If it was thought that a donor might have contributed the blood which caused the malaria attack, a number of thick blood films were taken which were later examined by Dr. T. C. Backhouse, of the School of Public Health and Tropical Medicine, Sydney.

Plasmodium Vivax Malaria.

The donors of blood for transfusion to two people who later developed *P. vivax* malaria were investigated.

CASE I.—The recipient was a woman, who had lived only in suburban Sydney. She received blood from four donors in the form of two transfusions on September 18 and 23, 1959. She developed an attack of *vivax* malaria on September 27, after she had been discharged from hospital.

The blood donor who contributed the infected blood (blood taken on September 14, transfusion on September 18) was found to be a soldier who had served in Malaya and returned to Australia in October, 1957. He had continued with suppressive "Paludrine" medication for a period and had then stopped taking it. In the period from April to June, 1959, he had been engaged in manœuvres in the Mackay (Queensland) area, when "Paludrine" was again used as a training procedure. He stated that he had had no attack of malaria since his return from Malaya, and none while serving in that country. A few days after making the blood donation he himself developed an attack of malaria. He was admitted to hospital on September 21, and trophozoites of *P. vivax* were found in his blood.

Of the other three donors, one was a soldier returned from Malaya in June, 1958, who had not had malaria; the other two had not been in malarious areas.

CASE II.—The recipient was a woman who had received two transfusions of packed red blood cells from four donors for anæmia late in pregnancy. The transfusions were given on September 26, 1957 (three donors), and September 27 (one donor). On October 3 she developed fever, and was admitted to hospital suspected of having pyelitis. During the subsequent eight days a tertian fever chart was recorded, and on October 14 trophozoites of *P. vivax* were found when a blood film was being examined to make a differential white cell count. She had not been north of Brisbane.

Three of the donors were resident in one of Her Majesty's gaols when they had made their blood donation. Two who were seen at Long Bay Gaol had not been in a malarious area. The third had been moved to Maitland Gaol, where he was interrogated by the visiting medical officer. He had been in New Guinea and returned to Australia about October, 1956. He had had malaria; but thick blood films taken in November, 1957, showed no malaria parasites. The fourth donor was a retired seaman who, although he had made many voyages in the Pacific, had never been in a malarious area.

In this case the third donor was assumed to have contributed the infected blood, although malaria parasites could not be demonstrated at the time of the examination. He was bled on September 24, 1957, and the transfusion of his packed red blood cells was given on September 26.

It is interesting to note in passing that gaol inmates appear to welcome the opportunity of giving blood; it is a change from the routine of everyday life.

P. Falciparum Malaria.

One case of accidental transfusion malaria due to infection with *P. falciparum* was investigated.

CASE III.—The recipient was a woman who received three blood transfusions (June 20 and 25, and July 8, 1958) from six donors in association with an operation for carcinoma. On July 2 her temperature rose, and on July 9 trophozoites and gametocytes of *P. falciparum* were found in her blood.

The donor responsible for giving the infected blood (bled on June 23, transfusion on June 25) had been a merchant seaman from 1942 to 1947. He had had an attack of fever in Trinidad in 1942. In the same year he was ashore at Rio de Janeiro, his ship having been torpedoed, and again he suffered from fever. In 1945 he was in West Africa and in the Persian Gulf, where he was treated for fever at Kuwait. In 1946 and 1947 he was in Dar es Salaam (Tanganyika), where he had frequent attacks of fever which he himself treated with quinine. In 1949 he went to New Guinea, and had an attack of fever at Rabaul in 1950.

In 1953 he was managing a plantation near Suangi on Guadalcanal (British Solomon Islands Protectorate). He had a severe attack of malaria in that year: he went to bed and treated himself with "Chloroquine" and brandy. He did not remember much of what happened during this illness. His house servants gave him "Chloroquine", but he vomited a great deal. They were also gloomy about the prognosis, and dug a hole to bury him. Since recovering from this attack of fever he had had headaches and fever which abated with aspirin only.

He returned to Sydney in April, 1957, where he discovered he had three tablets of "Chloroquine" in a packet. He took these at that time, to get rid of them. Since that time he had taken no antimalarial drug. In New Guinea and the Solomons he took no antimalarial drugs regularly.

TABLE I.

Details of Four Cases of Accidental Transfusion Malaria.

Case Number.	Parasite.	Country of Origin.	Period of Residence of Donor in Australia.	Period of Storage of Blood.	Incubation Period in Recipient.	Parasite Observed in Donor.
I	*P. vivax.*	Malaya.	23 months.	4 days.	9 days.	+
II	*P. vivax.*	New Guinea.	11 months.	2 days.	7 days.	−
III	*P. falciparum.*	British Solomons.	14 months.	2 days.	7 days.	+
IV	*P. malariæ.*	Algeria.	19 years.	5 days.	43 days.	+

Thick blood films were made from this donor on July 13, 1958, and trophozoites of *P. falciparum* were found at a density of about one per cubic millimetre. This was 14 months after his return from the Solomons, and he had not been in a malarious area in that period. He was also found to have an enlarged, firm spleen, palpable at the costal margin at full inspiration (Hackett Size I).

This donor had also given blood in March, 1958. The recipient in this case was a child with acute myeloid leukæmia, who was transfused with packed red blood cells from the present donor and one other. This child died 10 days later (after discharge from hospital), but there appeared to be nothing suggestive of malaria according to the honorary medical officer.

P. Malariæ Malaria.

One case of accidental transfusion malaria due to infection with *P. malariæ* was investigated.

CASE IV.—This patient was a woman with Korsakow's psychosis. She was examined in consultation concerning the possibility of some of her clinical features being due to a *malariæ* (quartan) malaria attack which had developed while she was in hospital. Owing to her mental condition she could not answer questions concerning her past movements, and information on this subject was not available from people who knew her. As *malariæ* infections are uncommon, the possibility of transfusion malaria was considered. It was found that she had received a blood transfusion in another hospital (June 19, 1956) with blood from two donors. She subsequently developed a urinary infection which responded to treatment with antibiotics. On August 1, she had pyrexia, which in subsequent days was seen to be quartan in type. On August 14 the significance of this quartan fever was realized, and a blood film demonstrated trophozoites of *P. malariæ*. It was subsequently ascertained that the patient was born in the Newcastle district (New South Wales) and had not been outside Australia.

The two donors of blood for this woman's transfusion were National Service trainees at the time of their donation. Their civilian addresses were obtained from the Deputy

Director of Medical Services' office in Sydney. One had never been outside New South Wales. The other was born of Maltese parents at Bône, Department of Constantine, Algeria, in 1936. He came to Australia when he was nine months old. In the years 1937 to 1940 he had lived at Innisfail in North Queensland, and then came to suburban Sydney where he had lived ever since. Neither he nor his mother could recall any fever attacks which might have been malaria, although his mother stated that she had had malaria at Bône. The spleen of this donor was not palpable; but examination of numerous thick blood films (taken on September 2, 1956) showed very scanty trophozoites including a mature schizont of *P. malariæ* (blood donation June 14, 1956).

The relevant details of these cases are presented in summary form in Table I.

Discussion.

There can be no doubt that in the cases reported here malaria was acquired as the result of blood transfusion. None of the recipients had been outside Australia, and in recent years endemic malaria (due to *P. vivax*) has occurred only in a small area of the Northern Territory. None of these recipients had been in that area. Admittedly, occasional local transmission has been reported in the southern parts of Australia, even in suburban Sydney; but in three of the cases malaria parasites were demonstrated in the donors, and in the fourth the history gives sufficient evidence to accept his blood as containing parasites at the time of the donation.

Little comment is necessary concerning the donors in the *vivax* cases. One was himself incubating an attack at the time of the donation. His infection was apparently acquired in Malaya, and it was almost two years since his return from that country. It is much less likely that he had been infected during manœuvres in the Mackay (Queensland) area. A period of 22 months from cessation of suppression to the onset of the first attack of *vivax* malaria is quite long; but similar findings were reported in soldiers returning home from the war in the South-West Pacific.

The other donor of *vivax* malarial blood showed no malaria parasites at the time of investigation. This is by no means surprising, in view of the cyclical activity of *vivax* malaria in man. The origin of his infection was in New Guinea, whence he had returned 11 months before making the donation. He had a history of malaria.

On the face of it, the donor of the blood containing *P. falciparum* presents a rather surprising story. Here was a man, born in England, who had a severe attack of malaria in the British Solomon Islands, and 14 months after returning to Australia still had *P. falciparum* circulating in his blood. Despite this he was quite well; indeed, he was a fit blood donor. This is the response that may be seen in indigenous people in areas where malaria is endemic; it is an unusual response in a European in the South-West Pacific. The explanation probably lies in this donor's previous experience with malaria. In the period 1942 to 1947 he had fever attacks in the West Indies, South America, the Persian Gulf and East Africa. These attacks, plus his attack in the Solomons, had rendered him tolerant to *falciparum* malaria, so that he could appear well (his red blood cells numbered 4,500,000 per cubic millimetre, his hæmoglobin value was 13·6 grammes per 100 ml.) when examined 15 months after leaving a malarious area, despite the presence of *P. falciparum* trophozoites in his blood.

The donor of the blood containing *P. malariæ* was apparently infected in Algeria in 1936. At Bône, where he lived as an infant, the spleen rate in the local people in 1934 was 17% (Ambialet, 1935), and in the Department of Constantine (in which Bône is situated) 5% of children aged under one year were found to be infected with *P. malariæ* in 1939 (*Institut Pasteur d'Algérie*, 1949). It is apparent, therefore, that this donor could quite easily have acquired his infection in Algeria. In his early years in Australia he lived in the Innisfail district at Goondi. Breinl (1919), in earlier years, reported that he had seen one case of quartan malaria from the Mourilyan district, which is not far from Goondi. However, it is much more likely that this donor's infection had been acquired in Algeria. When he first gave blood for transfusion 19 years later it contained *P. malariæ*. This is by no means an unusual story in infections with this parasite.

As many soldiers returned from service in New Guinea regularly make blood donations to the Transfusion Service, it is interesting to inquire into the incidence of *P. malariæ* infections which may be expected in these soldiers. The over-all incidence of this species in the 100,000 Australian army individual troops who suffered from malaria acquired in New Guinea is not known; but it has been possible to locate a number of reports in which the incidence of *malariæ* infections is recorded.

In the Milne Bay area of New Guinea, Lovell (1943) reported two of 961 malaria cases as due to *P. malariæ*. In Port Moresby, Williams (1943) found that seven of 1000 malaria cases were due to infection with this species. Again, 11 of 9802 malaria cases recorded for New Guinea (army hygiene records) were due to *P. malariæ*. At Toowoomba, Lancaster found two infections due to this species in a total of 716 cases. As an approximation, these cases may be added together to give 22 *P. malariæ* infections in a total of 12,479 cases of malaria in army troops, an incidence of 0·18%.

It is possible that one or more individuals with *P. malariæ* infections may have been recorded more than once in this series; but it is more likely that this percentage should be applied to the 100,000 individuals who acquired overt malaria rather than to the 200,000 admissions for malaria. This gives a figure of 180 individuals who suffered from overt *malariæ* infections.

This figure must, however, be taken as an approximation. A number of persons may have been infected without clinical symptoms (as was apparently the case with the donor under discussion). Again, the species diagnosis may easily have been missed in a very busy laboratory. However, the number of cases is very small.

The significance of these cases in regard to the acceptance as blood donors of people who have lived in malarious areas requires some discussion. The matter could easily be dismissed by refusing anyone who has been resident in a malarious country. However, in Sydney this would exclude quite an appreciable number of people who wish to become voluntary donors. This was especially the case in the years after World War II, when soldiers who had had service in New Guinea wished to make blood donations.

Thus some compromise had to be made, and the Transfusion Service was advised to question all donors about past residence in a malarious country. If the prospective donor had resided in a malarious country, he was accepted if it was two years or more since he had been in a malarious country and he had had no malaria attacks for at least two years without taking a suppressive (Walsh and Ward, 1957). This approach proved to be effective: there were no cases of accidental transfusion malaria. In more recent years there has been an increasing demand for blood donors, and at the same time the Korean War, the employment of Australian troops in Malaya, and the much-increased global mobility of Australians in general led to an increasing number of prospective donors being rejected on these criteria. The Transfusion Service was then advised to reduce the period of freedom from malaria attacks to one year, with the reservation that an occasional case of accidental transfusion malaria might appear.

All four of the donors involved in the cases reported here show more or less unusual features. The donor in Case I (*vivax* malaria) showed no clinical activity until 23 months after his return from Malaya — a rather long period. In Case II (*vivax* malaria) the donor misled the Transfusion Service with false statements about his malaria history and had not completed the specified period of one year. The donor in Case II (*falciparum* malaria) was quite unusual, as has already been indicated; he, too,

had made a previous blood donation before the stipulated year had expired. Abdominal palpation would have revealed his enlarged spleen. The donor in Case IV (*malariæ* malaria) represents one of a large group of cases of accidental transfusion malaria which have been reported in many parts of the world. Such cases must inevitably occur, albeit rarely, when donors are used who have been in malarious areas, even if their sojourn has ended up to 20 or 40 years before they make their donation.

With these points in view, there appears to be no valid reason for altering the criteria at present in use for accepting or rejecting prospective blood donors who have been in malarious countries. In the four years 1956-1959, when these four cases occurred, 217,948 bottles of blood were issued from the Central Headquarters of the Blood Transfusion Service. There is an important corollary to this opinion: medical practitioners who use blood for transfusions must be aware of the possibility of the rare complication of accidental transfusion malaria. In all cases in which pyrexia follows blood transfusion, the possibility of malaria being the cause should be considered, and this should be considered early.

If diagnosis is not made quickly and appropriate therapy administered, the malaria attack may well reduce the patient to a condition worse than before the transfusion was given. Delay in diagnosis of *falciparum* malaria may prove fatal. In the cases recorded here, the periods between onset of pyrexia and diagnosis of malaria were as follows: for the *vivax* cases, eight days (only the last two of these days in hospital) and 12 days; for the *falciparum* case, seven days; and for the *malariæ* case, 13 days.

Further, when acute malaria is diagnosed in a patient and natural infection seems unlikely, the possibility of infection by means of a blood transfusion should be investigated. This transfusion may have been given up to three months before in *malariæ* (quartan) infections; in the case presented here, malaria was diagnosed 56 days after the blood transfusion which caused the infection.

Summary.

1. The 16 blood donors concerned in four cases of accidental transfusion malaria have been investigated.

2. Four of these donors were identified as those who had supplied the blood which caused the infection in the recipients.

3. The parasite species involved were: *P. vivax* (two cases), *P. falciparum* (one case) and *P. malariæ* (one case).

4. As far as is known, these four were the only cases of accidental transfusion malaria in the period 1956-1959, when 217,948 bottles of blood were issued from the Central Headquarters of the Red Cross Blood Transfusion Service in Sydney.

5. Despite the low incidence of this complication of blood transfusion, the possibility of malaria must always be entertained when there is pyrexia in a patient who has recently received a blood transfusion.

6. Some previously unpublished figures concerning the incidence of *P. malariæ* infection in Australian army personnel in World War II are presented.

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APLASTIC ANÆMIA FOLLOWING THE ADMINISTRATION OF PHENYLBUTAZONE.

By G. S. HALE, M.D., M.R.A.C.P., AND G. C. DE GRUCHY, M.D., F.R.A.C.P., M.R.C.P., M.C.P.A.,

From the Department of Medicine, St. Vincent's Hospital, Melbourne.

IT is well known that agranulocytosis is an important and not uncommon complication of phenylbutazone ("Butazolidin") administration. Thus Mauer (1955), in a review of the literature, found reports of 25 cases of agranulocytosis, 10 of which were fatal. On the other hand, the occurrence of aplastic anæmia is much less widely recognized, and to our knowledge only five cases have been previously reported.

The purpose of this paper is to review the literature briefly and to report four cases of aplastic anæmia which followed the administration of phenylbutazone.

There are five cases of aplastic anæmia following phenylbutazone reported in the literature. Some of the more important features of these cases are listed in Table I.

Leonard (1953) recorded the case of a woman, aged 64 years, with rheumatoid arthritis; she received a course of phenylbutazone in a dosage of 600 mg. per day for 14 days. One week after the commencement of treatment her doctor noted that she had a purpuric rash on the legs, and during the course there was slight bleeding from the nose and mouth. On the fourteenth day of treatment she was found to be severely anæmic (hæmoglobin value 3·8 grammes per 100 ml.) and breathless, and was admitted to hospital as an emergency. However, this woman had received a course of gold injections (total 0·44 gramme) commencing 11 weeks and finishing seven weeks before the onset of the purpuric rash. Gold is a well-recognized cause of aplastic anæmia, and there is commonly a latent period of several weeks to several months between the commencement of therapy and the onset of symptoms of marrow depression. Thus it seems more likely that the aplastic anæmia was due to the gold than to the phenylbutazone, especially as bleeding symptoms occurred within seven days or less of the commencement of phenylbutazone administration, and as the patient was severely anæmic within 14 days in the absence of obvious hæmorrhage or hæmolysis.

Venning (1957) reported the case of a woman, aged 55 years, who was treated with phenylbutazone for "pains in the back and various joints". She had taken a dose of between 300 and 600 mg. daily for about 20 months, at the end of which time she complained of bruising easily and of a rash "like blood blisters", and she began to feel weak. She was admitted to hospital about two months later as an emergency, with severe anæmia (hæmoglobin value 6 grammes per 100 ml.). She did not respond to a course of cortisone lasting about seven weeks; despite blood transfusion (27 pints) she died as the result of hæmorrhage in the alimentary tract about four months after her admission to hospital. The only other drug she had taken was thyroid. There is no record of other toxic manifestations of phenylbutazone. It seems most probable that the aplasia in this case was due to the phenylbutazone.

Hillemand, Isch-Wall and Delavierre (1958) record the case of a woman, aged 71 years, with chronic polyarthritis; she took an average daily dose of 200 mg. of phenylbutazone for 11 months; during this period she

TABLE I.

Cases in the Literature—Clinical Features.

Author.	Sex.	Age. (Years.)	Disorder Treated.	Dose.	Interval Between Last Dose and First Symptoms.	Other Toxic Manifestations of Phenylbutazone.	Other Drugs Taken.	Treatment.	Outcome.
Leonard (1953).	F.	64	Rheumatoid arthritis.	600 mg. daily for 2 weeks; total: 8·4 grammes.	Still taking drug.	Nil.	Course of gold injections (total 0·44 gramme) commenced 11 weeks and finished 7 weeks before onset of symptoms.	Blood transfusion.	Death; cerebral hæmorrhage, pulmonary congestion and hæmorrhage.
Venning (1957).	F.	55	Back and joint pains.	Average dose probably 400 mg. daily for 20 months; approximate total: 200 grammes.	Still taking drug.	Nil.	Thyroid, 2 grains daily, 2 years.	Blood transfusion, cortisone.	Death; gastrointestinal hæmorrhage.
Hillemand *et alii* (1958).	F.	71	Chronic polyarthritis.	200 mg. daily for 11 months; approximate total: 66 grammes.	Still taking drug.	Nil.	Nil.	Blood transfusion, cortisone.	Death from "multiple hæmorrhages".
Mohler and Leavell (1958).	F.	55	Rheumatoid arthritis.	——	——	—	——	Prednisone.	Full recovery.
McCombs (1958).	F.	80	Pain in shoulders, wrists and hands.	Large amounts over a period of nine months.	Still taking drug.	Nil.	Nil.	Blood transfusion, ACTH, cortisone.	Death from multiple hæmorrhages and pneumonia.

did not take any other medicine. After 11 months' therapy, she complained of purpura, headaches and giddiness; she stopped taking phenylbutazone, but her symptoms became worse, and she also noted bruises, epistaxis, hæmaturia and the passage of bright blood per rectum. She received six transfusions, but died three months after the onset of symptoms from "multiple hæmorrhages". There is no record of any other toxic manifestation of phenylbutazone. It seems probable that the aplasia in this case was due to the phenylbutazone.

Mohler and Leavell (1958), in an analysis of 50 cases of aplastic anæmia, briefly record a case of aplastic anæmia in a woman, aged 55 years, which they attributed to phenylbutazone. Her illness lasted 16 months and was followed by complete recovery. Details of this case are not given; it is stated that they will be given in a separate report.

McCombs (1958) reported the case of a woman, aged 80 years, who took phenylbutazone "on and off" for nine months before the onset of symptoms, when she developed the clinical and hæmatological features of aplastic anæmia. The patient died of hæmorrhage and pneumonia about four weeks after the onset of symptoms; autopsy confirmed the diagnosis of aplastic anæmia. The exact dosage of phenylbutazone could not be determined; but it was considered that the patient had taken large amounts of the drug over a period of nine months. There was no mention of other manifestations of phenylbutazone toxicity.

Reports of Cases.

CASE I.—A housewife, aged 58 years, was admitted to hospital on April 5, 1956, complaining of fatigue present for six months, of spontaneous bruising present for three weeks, and of a sore throat of one week's duration; one week previously she had had an epistaxis. Examination of the patient on her admission to hospital showed her to be a pale, middle-aged woman, with multiple petechiæ and bruises on the skin of the trunk and limbs; the tourniquet test gave a strongly positive result. The temperature was 100° F.; the throat was injected, but not ulcerated. The spleen and liver were not palpable, and there was no palpable lymph-node enlargement and no sternal tenderness. Blood examination revealed pancytopenia with severe neutropenia and thrombocytopenia, and a raised erythrocyte sedimentation rate (Table II). In the blood-film the red cells showed moderate anisocytosis with little poikilocytosis. Marrow aspirated from the sternum was aplastic, with empty marrow fragments and only a few normoblasts and developing myeloid cells in the cell trails.

For the past 25 years she had suffered from rheumatoid arthritis, which had caused permanent deformity of both hands. Her treatment included a course of gold injections ten years previously, and ACTH and cortisone two years previously. She had received a course of phenylbutazone (100 to 200 mg. per day) for a period of 12 months, the course having ceased four months before her admission to hospital, and two months after the onset of fatigue. On ceasing the phenylbutazone, she was given a course of prednisone (dose uncertain) for approximately three months; the course ceased three weeks prior to her admission. There was no history of a rash or of any other manifestation of phenylbutazone toxicity. One week before her admission she had been given a transfusion of two pints of blood at another hospital.

Her immediate treatment consisted of the administration of procaine penicillin for five days in a dose of 600,000 units twice a day, and ACTH gel given intramuscularly in a dose of 20 units twice a day for 11 days. After 11 days she was given a transfusion of two pints of blood, ACTH was ceased and a course of prednisone was commenced, the dosage being 60 mg. per day for 16 days, followed by decreasing daily doses over the next seven days. The administration of the adrenocortical steroids resulted in a slight improvement in the blood picture, but not in her clinical condition; hæmorrhagic manifestations persisted, and paronychia developed. Because it was considered that her life was in danger from bleeding and possibly from infection, splenectomy was performed on May 15, cortisone being administered in decreasing doses for one day before and seven days after operation. Splenectomy was followed immediately by a moderate improvement in the blood picture, particularly in platelets and neutrophils; on the eighteenth day after operation a course of ACTH gel ("Corticotrophin Z") in a dosage of 40 units per day was commenced; this was folowed by a prompt increase in all three formed elements. Nine weeks after splenectomy the blood picture was within the normal range except for slight neutropenia (1700 per cubic millimetre), and two years after the operation all three formed elements were within the normal range.

A detailed account of the response to adrenocortical steroids and splenectomy in this case is being included in a separate report (de Gruchy and Hale).

The only other agent known to cause aplastic anæmia which this patient had received was gold, given 10 years previously. It seems probable that her aplastic anæmia resulted from the phenylbutazone. There was definite evidence of response to the course of adrenocortical steroid hormone therapy given after splenectomy.

CASE II.—A housewife, aged 60 years, was admitted to hospital on May 2, 1957, with a three weeks' illness beginning with a sore throat, which her doctor diagnosed

TABLE II.
Hæmatological Findings at Presentation.

Case Number.	Hæmoglobin Value. (Grammes per 100 Millilitres.)	Total Number of Leucocytes (per Cubic Millimetre).	Neutrophils (per Cubic Millimetre).	Platelets (per Cubic Millimetre).	Reticulocytes.	Erythrocyte Sedimentation Rate (Westergren), Millimetres per per Hour.	Bone Marrow.
I	8·1	1500	50	10,000	0·1%	127 (infection present)	Aplastic.
II	8·8	1000	300	12,000	0·1%	138 (infection absent)	Aplastic.
III	15·7 (post-transfusion)	3000	50	7000	0%	30 (infection absent)	Aplastic.
IV	6·7	1050	350	20,000	0·7%	75 (infection absent)	Aplastic.

as acute pharyngitis, and for which she was given a single injection of procaine penicillin (900,000 units). Her throat improved over the next two days but she then noticed the sudden onset of spontaneous bruising, a periorbital hæmatoma and bruising of the limbs appearing almost overnight. Over the next two weeks, the bruising persisted and she became lethargic. One week before her admission she developed epigastric pain and melæna which subsided after three days.

Examination of the patient on her admission to hospital showed her to be a pale woman with a normal temperature. The outstanding feature was the presence of multiple bruises over the whole of the body; there were also a few petechiæ on the legs and buccal mucosa; the tourniquet test produced a strongly positive result. There was no hepatomegaly, splenomegaly or bone tenderness. Blood examination revealed pancytopenia with severe neutropenia and thrombocytopenia, and a raised erythrocyte sedimentation rate in the absence of obvious infection (Table II). Sternal marrow aspiration yielded no fragments; a second aspiration from the iliac crest yielded a few empty marrow fragments with very few cells in the cell trails; the few myeloid cells present showed heavy toxic granulation.

The patient had suffered from pain in the hands and left foot for 12 months prior to her admission to hospital, and X-ray examination of the left foot revealed subluxation of the mid-tarsal joint. For a period of four months prior to the onset of symptoms of aplastic anæmia, she had taken, with some relief, phenylbutazone tablets in a dose of 200 mg. twice a day; on the advice of her doctor she ceased taking the tablets after the onset of the bruising. At no time did she have a rash or any other symptoms of phenylbutazone toxicity. After ceasing the phenylbutazone (that is, after the onset of bruising), she took a course of salicylamide tablets, one tablet three times a day for one week. During the previous 12 months she had also taken œstrogen and amphetamine sulphate tablets.

She was treated with ACTH gel, and a significant improvement in the hæmatological picture was noted by the end of two weeks; in addition, she did not develop any fresh bruises or other new symptoms. The initial dose of ACTH gel given intramuscularly was 40 units per day for 10 days; this was reduced to 20 units per day for eight weeks and then to 10 units per day for about six weeks. The initial rapid improvement was followed by a slow progressive improvement in the blood picture, until in December, 1959, all three formed elements were within normal limits. Apart from her painful foot she is clinically well. Two episodes of infection caused temporary depression of the hæmatological response.

The details of this patient's response to treatment are being included in a separate report (de Gruchy and Hale).

The only agents other than phenylbutazone taken before the onset of the aplasia were œstrogens and amphetamine sulphate; as these have not been reported as causing aplasia, it seems most likely that the aplasia was due to the phenylbutazone. There was definite evidence of response to adrenocortical steroid hormone therapy, and of the depressant effect of the intercurrent infection on the marrow response.

CASE III.—A housewife, aged 70 years, was admitted to hospital on July 2, 1957, complaining of weakness of five weeks' duration and of spontaneous bruising and petechiæ of two weeks' duration. She had consulted her doctor on the day when she first noticed the petechiæ, which were then localized to the feet. The doctor suspected a blood dyscrasia and immediately stopped the drugs she was taking at the time—namely, phenylbutazone and chlorpromazine. Nevertheless, the petechial rash became generalized, and "blood spots" developed in the mouth. Blood examination on June 21 gave the following information: hæmoglobin value, 11 grammes per 100 ml.; leucocytes, 4300 per cubic millimetre (neutrophils 1400 per cubic millimetre); bleeding time, 10 minutes; platelets scarce in the blood film. On June 31 her hæmoglobin value had fallen to 9 grammes per 100 ml., and she was transfused with four pints of blood; the neutrophils numbered 750 per cubic millimetre, and the platelets 50,000 per cubic millimetre.

Examination of the patient at the time of her admission to hospital showed her to be a pale elderly woman with petechiæ and bruises scattered over her trunk and limbs; the response to the tourniquet test was strongly positive. There was no lymph node enlargement, hepatomegaly, splenomegaly, bone tenderness or pyrexia. Sternal bone marrow aspiration biopsy yielded very hypoplastic marrow with hypoplastic fragments; the cell trails were markedly hypocellular and contained only an occasional normoblast, a few myeloid cells showing toxic changes and a few plasma cells and lymphocytes.

Since the age of 50 years the patient had suffered from rheumatoid arthritis, and for the last two years she had been given four courses of phenylbutazone. The average daily dose was 200 mg., and the total amount of the drug taken during two years was about 50 grammes. She had no sign of toxicity from the drug; there was no rash, and frequent blood examinations during the first three months of therapy were reported as having given normal results. The relief that she experienced from the phenylbutazone was such that she stated that she "could not carry on without the tablets". Other treatment for her arthritis had included salicylates and gold injections; no details of the dose or effects of the gold could be obtained, but it is certain that she had had none since 1953—four years before her present illness. She had also been given insulin for diabetes mellitus from November, 1956, to February, 1957, and chlorpromazine for mental depression in a dose of 75 mg. per day from April, 1957, to June, 1957.

Treatment with prednisone was begun on July 2 in a dose of 15 mg. per day for two weeks. On July 17, she was given a further transfusion of two pints of blood. After the transfusion her blood picture was as follows: hæmoglobin value, 15·7 grammes per 100 ml.; leucocyte count, 3000 per cubic millimetre (less than 50 neutrophils); reticulocyte cell count, 0%; platelets, 7000 per cubic millimetre; erythrocyte sedimentation rate, 30 mm. per hour. On this date the dose of prednisone was increased to 60 mg. per day. Death occurred suddenly on July 19, following melæna and the onset of signs suggesting an intracerebral hæmorrhage. An autopsy was not obtained.

This patient had been taking two drugs prior to her admission to hospital—namely, phenylbutazone and chlorpromazine. Chlorpromazine is a well-recognized cause of agranulocytosis, but the literature to date does not appear to contain any reports of aplastic anæmia with pancytopenia following chlorpromazine therapy. Nevertheless, the Council on Pharmacy and Chemistry of the American Medical Association (1956), reporting on cases that had come to their notice, stated that "the blood dyscrasia most often reported was agranulocytosis, although in many cases bone marrow studies revealed depression of other cellular elements as well as the granulocyte precursors". Thus it is possible that the

TABLE III.
Clinical Features.

Case Number.	Sex.	Age. (Years.)	Disorder Treated.	Dose.	Interval Between Last Dose and First Symptoms.	Other Toxic Manifestations of Phenylbutazone.	Other Drugs Taken.	Treatment.	Outcome.
I	F.	58	Rheumatoid arthritis.	100 200 mg. per day for 12 months continuously. Approximate total: 50 grammes.	Still taking drug.	Nil.	Cortisone. ACTH two years previously, gold injections 10 years previously, prednisone for 4 months before admission to hospital.	ACTH, prednisone, splenectomy.	Recovery.
II	F.	60	Arthralgia of hands; painful left foot (mid-tarsal subluxation).	400 mg. daily for 4 months continuously. Approximate total: 50 grammes.	Still taking drug.	Nil.	Œstrogens, amphetamine sulphate, salicylamide for 1 week after diagnosis, penicillin 3 weeks before diagnosis.	ACTH, antibiotics.	Recovery.
III	F.	70	Rheumatoid arthritis.	Four courses in 2 years, 200 mg. daily. Fourth course lasted 50 days. Approximate total: 50 grammes.	Still taking drug.	Nil.	Gold injections 4 years previously, salicylates intermittently, insulin 6 months previously, chlorpromazine for 3 months before admission.	Prednisone, blood transfusion, antibiotics.	Death; gastro-intestinal and cerebral hæmorrhage.
IV	M.	74	Osteoarthritis of right hip.	Average dose 200 mg. daily, continuously for 2 years. Approximate total: 150 grammes.	Still taking drug.	Nil.	Cod-liver oil.	ACTH, blood transfusion.	Death; gastro-intestinal and cerebral hæmorrhage.

aplasia in this case resulted from the chlorpromazine, although on the basis of probability it was more likely due to phenylbutazone. There was no evidence of response to adrenocortical steroid hormone therapy in this case; however, the dose of prednisone given initially was not adequate, and the patient died only two days after adequate doses were commenced.

CASE IV.—A retired carpenter, aged 74 years, was admitted to hospital on August 31, 1957, with a six weeks' history of intermittent bleeding from the nose, excessive bleeding from shaving cuts and progressive weakness. He had not had any serious illnesses in the past, but for some years had suffered from osteoarthritis of the right hip. Apart from pallor and a strongly positive response to the tourniquet test, there were no positive findings on physical examination. Because of the clinical severity of his anæmia, an emergency transfusion of two pints of blood was given in the casualty department. Blood examination after this transfusion showed severe pancytopenia (Table II), with a bleeding time of longer than 15 minutes; there was no clot retraction. Sternal marrow examination revealed aplastic marrow with empty marrow fragments, and only a few cells in the cell trails, the majority of which were lymphocytes and plasma cells. The occasional myelocytes and neutrophils seen showed coarse toxic granulation. Trephine biopsy of the iliac crest also showed marked marrow aplasia.

The patient was closely questioned about the possibility of exposure to a toxic ætiological agent. He admitted to having taken cod-liver oil and concentrated fruit juices, but denied taking any tablets. However, his relatives revealed the fact that he had taken phenylbutazone tablets for a period of two years. The dose was uncertain, as he had been able to obtain supplies of the drug without fresh prescriptions. His original prescription was for a two weeks' course of 200 mg. twice a day. Subsequently, as far as could be determined he took on an average one tablet per day until one to two weeks before his admission to hospital. It has been calculated that the total dose must have been at least 150 grammes.

The treatment given was as follows. On his admission to hospital he was given a course of ACTH gel, 20 units twice a day by intramuscular injection for three weeks. This was followed by some lessening of the bleeding tendency, with a decrease in the number and severity of the epistaxes; the result of the tourniquet test became less strongly positive, and the number of platelets rose temporarily from 20,000 to 40,000 per cubic millimetre. There was no change in the reticulocyte or leucocyte counts, but the sedimentation rate fell from 60 to 30 mm. in one hour. Two weeks after the commencement of therapy he developed an ulcerated throat and high fever, associated with a rise in sedimentation rate from 30 to 120 mm. per hour. These responded to the administration of penicillin and streptomycin for one week. He was discharged after five weeks in hospital, during which time he had received 13 pints of blood. Three weeks later he was readmitted because of anæmia due to recurrent nose bleeds, and received 10 pints of blood over a period of three weeks. He was then discharged, but was readmitted for the third time nine days later with profuse epistaxis and melæna. He received five pints of blood, but developed a cerebral hæmorrhage from which he died 12 hours later—that is, about 17 weeks after the onset of symptoms. Permission for an autopsy was not obtained.

The only drug known to cause aplasia taken by the patient was phenylbutazone, and it therefore seems probable that the aplasia was due to this drug. It is of interest that a history of ingestion of the drug could not be obtained from the patient, but required questioning of the relatives, and that the drug was being taken without medical supervision. There was no significant response to steroids given in an adequate dose for a period of three weeks, apart from a temporary symptomatic reduction in bleeding and a slight increase in the platelet count.

Discussion.

It is not possible to prove definitely a causal relationship between the administration of a drug and the occurrence of aplastic anæmia which follows the administration of that drug. Nevertheless, when aplastic anæmia develops on a number of occasions after the administration of a particular drug, it is reasonable to assume that the drug causes aplastic anæmia, especially when the patients have not been exposed to any other toxic agent. In three of our cases (Cases I, II and IV) no other drug thought to cause aplasia had recently been administered, and it seems probable that the aplasia in these cases was due to the phenylbutazone. In Case III chlorpromazine had also been administered, and it is possible that the aplasia was due to this drug rather than to the phenylbutazone. Of the cases in the literature, those of Venning (1957), Hillemand *et alii* (1958), Mohler and Leavell (1958) and McCombs (1958) are probably due to phenylbutazone; that of Leonard (1953) is more likely to be due to the gold that the patient had received. Thus, of a total of nine cases (our four cases and five cases in the literature) of aplastic anæmia following phenylbutazone administration, it seems that the aplastic anæmia was probably due to the phenylbutazone in seven and possibly due to it in one.

It is of interest to note the difference between the relation of time of onset and of dose in agranulocytosis following phenylbutazone and the cases of aplastic anæmia reviewed in this paper. Mauer (1955) found, in the reported cases of agranulocytosis, that the agranulocytosis had occurred at various times from the seventh to the sixtieth day of treatment, and that there was no

apparent relation to dosage, which ranged from 3·5 to 39·2 grammes. By contrast, it would seem that aplasia had occurred only after prolonged administration, when relatively large total amounts of the drug had been taken. The doses in our Cases I to IV were 50, 50, 50 and 150 grammes respectively, and the periods of administration 12, 4, 24 and 24 months. Of the two cases in the literature with details of dosage, those of Venning (1957) and Hillemand *et alii* (1958), the total doses were 208 and 66 grammes respectively, and the periods of administration 20 and 11 months; McCombs's (1958) patient had taken the drug in large amounts over a period of nine months. The average daily doses in our cases and those in the literature (Tables I and III) ranged from 200 to 400 mg. per day.

It is also of interest to note that none of our patients exhibited any of the other recognized toxic manifestations of phenylbutazone, and that they were not recorded as occurring in the cases described in the literature. The absence of a rash is particularly noteworthy. Mauer (1955) found that rashes were noted in almost all the cases in which agranulocytosis developed in the first four weeks of treatment, the skin reactions preceding blood changes by intervals of 4 to 15 days; on the other hand, when agranulocytosis developed after the fourth week it was not preceded by a skin rash. As was pointed out above, all the reported cases of aplasia have occurred long after the fourth week of administration.

Like all aplastic anæmias, the disorder is serious, death occurring in five of the eight acceptable cases (Tables I and III). Recovery was definitely hastened in our Cases I and II by the administration of adrenocortical steroids, and was possibly helped by splenectomy in Case I.

Summary.

Four cases of aplastic anæmia which followed the administration of phenylbutazone are described.

Previous cases described in the literature are reviewed.

The relation between duration of administration and dosage is discussed. It is noted that, in general, aplastic anæmia occurred after prolonged administration, in contrast with agranulocytosis, which often occurs after relatively short administration.

The absence of other symptoms of phenylbutazone toxicity is noted.

Of a total of eight patients, five died and three recovered. In two of our cases which ended in recovery, the administration of adrenocortical steroids resulted in a prompt and sustained improvement in the blood counts.

In view of the extensive use of phenylbutazone and the small number of cases of aplastic anæmia reported, the incidence must be low. Nevertheless, because of the serious nature of the complication, practitioners should be aware of its occurrence.

Acknowledgements.

We wish to thank Dr. Kate Mackay for permission to publish details of Case III.

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STAGED REPAIR OF MASSIVE INCISIONAL HERNIÆ.[1]

By EDWARD WILSON, M.D., M.S., M.Sc., F.R.C.S. (England), F.R.C.S. (Edinburgh), F.R.A.C.S., F.A.C.S., M.R.A.C.P.,
Sydney.

WITH some massive incisional herniæ the problems of treatment are insurmountable. Others may appear so at first; yet, by the adoption of various devices, cure is not impossible. Such a device is staging the repair of a hernia, so that at each stage the repair is proceeded with only so far as that is possible without producing undue tension. Then, except after the final stage, the unrepaired portion of the hernia acts as a safety-valve and limits the rise of intraabdominal pressure.

Herniation may occur through any previous incision; but herniæ of the chest wall, of the lumbar region or of the perineum are not included in this paper, nor are herniæ which have followed operations on distant sites. Examples of the latter are a right inguinal hernia after appendicectomy, an inguinal hernia following operation on a femoral hernia, and its opposite—a femoral hernia following operation on an inguinal hernia. In other words, this paper is concerned only with herniæ which arise at the sites of incisions in the abdominal wall.

Incisional herniæ differ greatly in their contents; but in the assessment of a hernia before operation, it is often more important to determine the following: (i) the extent of the loss of muscle and fascia; (ii) the type of incision responsible for the hernia; (iii) the proximity of the hernia to bony structures; (iv) the size of the hernial orifice; (v) the degree of tension in the hernia on coughing or straining, and whether the edges of the hernial orifice then come together; (vi) the degree of incapacity produced by the hernia; (vii) whether the symptoms are relieved by wearing an abdominal belt or by lying down; (viii) the nutritional state of the patient.

Few patients suffering from an incisional hernia come for treatment whilst the hernia is still small. In many cases the minor discomforts of the hernia are tolerated until other disabilities arise. Then the total burden becomes overwhelming, and cure of the hernia is demanded. Frequently some degree of cardiac insufficiency or increasing obesity is the last straw which is the immediate cause of the visit to the surgeon. In the past this delay of the operation until the hernia is no longer uncomplicated has been insufficiently stressed.

While they persist, contraindications to the operative repair of incisional herniæ are (i) ascites; (ii) persistent sepsis; (iii) persistent hydatidosis; (iv) wide separation of the attachments of the recti muscles to the ribs in patients with symptoms relieved by wearing a support; (v) a short life expectancy and slight symptoms; (vi) pregnancy; (vii) an intraabdominal tumour.

If the anæsthetist is experienced in the care of bad-risk patients, the poor general condition of a patient may not be a contraindication to operation. In patients with gross cardiac insufficiency the operation may be performed with the patient in the sitting position. Also, when the hernia is very large, its size is a challenge to the surgeon, but is not necessarily a contraindication to repair.

Prophylaxis.

The choice of incisions for abdominal surgery is still partly a matter of personal preference. Although paramedian incisions have enjoyed great popularity, there is

[1] From the Department of Surgery, Sydney Hospital, and the Department of Surgery, St. George Hospital, Sydney.

no proof that the rectus muscle is effective in preventing the formation of a hernia or in preventing enlargement of a hernia once it has commenced (Taylor, 1948). A mid-line abdominal wound presents a single, strong layer for repair instead of the two weak layers provided by a paramedian wound, and my personal preference is for the former.

Incidentally, the operation of oversewing a perforated peptic ulcer is the abdominal operation most often followed by an incisional hernia. For that reason the length of the incision for this operation should be kept small, 1·5 in. usually being quite adequate.

The initial repair of an abdominal incision and the care of the patient afterwards, especially during recovery from the anæsthetic, do not come within the scope of this paper, although they are important in the genesis of incisional herniæ.

Preoperative Preparation.

Before operation on an incisional hernia, the preparation of a patient should include the following measures: (i) special care of the skin if it is not healthy; (ii) improvement of the function of the myocardium; (iii) reduction

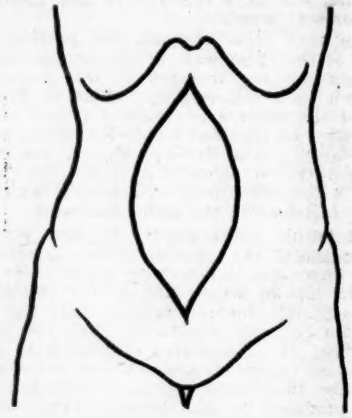

FIGURE I.
The orifice of an incisional hernia which required a two-staged repair. This hernia had followed a paramedian incision.

of obesity. Apart from the general effects, a lessening of obesity means a decrease in the intraabdominal fat and a corresponding decrease in the intraabdominal pressure after operation.

Any cause of a forceful cough, such as chronic bronchitis or sinusitis, will be actively treated, and all patients should learn to cough quietly and without greatly increasing the intraabdominal pressure. Smoking should be given up permanently or for as long as possible.

In elderly men the preoperative investigations include an assessment of the renal function and degree of prostatic obstruction. It is an advantage if the patient has already had a prostatectomy; otherwise, if there is any indication of even mild prostatic obstruction, this should be remedied first.

One method of reducing the intraabdominal pressure after operation is the previous induction of progressive pneumoperitoneum (Moreno, 1947). Prior to the operation, the hernia is compressed with adhesive strapping and the abdominal muscles are gradually stretched, so that repair of the hernia without tension is facilitated. A similar effect is produced by a very large abdominal tumour, and even though the muscles have been stretched and are weakened, closure of the wound after removal of such a tumour is usually so easy that an incisional hernia is unlikely.

The use in bed of the urinal and bed-pan is practised before operation, so that there will be less straining later. Also, the patient should practise rolling on to the side and flexing the trunk in this position before sitting up.

Operation.

If a hernia is closed under much tension, the suture line in the aponeurosis will, of course, break down, and the recurrent hernia may even be larger than the original. Instead of taking this risk, it is proposed that a large hernial defect should be repaired as far as possible without tension at the first stage, while operation on the remainder

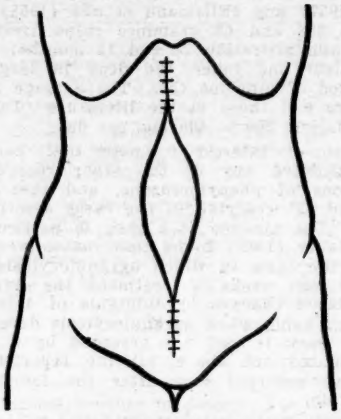

FIGURE II.
The hernial orifice shown in Figure I has been partly closed at each end. Closure of the residual defect was delayed until healing of this stage was firm.

is postponed for another one or more months (Figures I to V). At the second stage the smaller defect may be repaired or further reduced with much less tension, either directly or by the use of an implant. If necessary, closure is completed at a third stage. The alternative in a difficult case is attempting, with little chance of success, to obtain complete closure at a single operation.

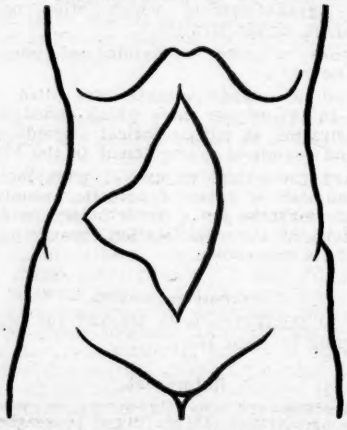

FIGURE III.
A large, irregular gap in the abdominal wall following a T-shaped incision. A two-staged repair was required.

After the first stage of its operative repair, the unrepaired portion of the hernia acts as a safety valve, and this reduces any sudden rise in intraabdominal pressure which may damage the suture line.

For a similar reason, if two separate incisional herniæ coexist, it is wise to delay repair of the second until the first is firmly healed.

During the past 10 years, the operative repair of 12 massive herniæ with gross loss of tissue has been

staged. In 10 of the cases the repair was in two stages, whereas three stages were necessary in the other two cases. In no case so far has there been a recurrence.

When the operative repair of a hernia is in stages, the same general principles applicable to the repair of other herniæ still pertain. Thus it is necessary to achieve the following objects:

1. Avoid tension. This is probably the single most important point in the repair of a hernia. Apart from leading to recurrences, tension may cause sloughing of the edges of the aponeurosis. The sloughs then act as irritant foreign bodies and cause infection and sinus formation.
2. Clear the fat from that portion of the aponeurosis used in the repair.
3. Repair the original layers as far as possible.
4. Overlap the edges of the defect if possible.
5. Eliminate any dead space.
6. Obtain complete hæmostasis.
7. Use many fine, interrupted, non-absorbable sutures.
8. Close an abdominal hernia transversely rather than vertically if there is any choice.

In addition, the urepaired portion should not be left too small, lest bowel becomes caught in it.

Operative measures to reduce the intraabdominal pressure include phrenic crush (Touroff, 1954), relaxing incisions in the abdominal muscles and fasciæ, and so-called tension sutures; but these measures are not required when the repair is staged.

When an implant is used, it should, whatever its type, be firmly fixed, it should be covered with a good layer of subcutaneous tissue and skin, and all grease should be removed from it. Complete hæmostasis is essential, and if there is any exudation of serum, this should be aspirated. Such a collection of serum or the presence of grease will greatly delay the proliferation of fibroblasts around the implant.

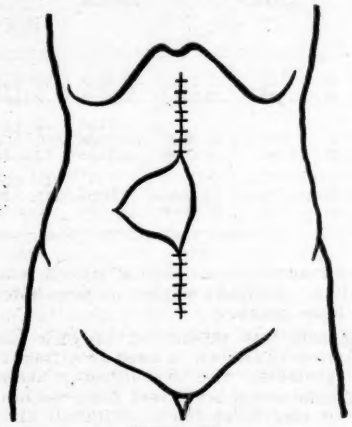

FIGURE IV.
Part of the hernial orifice shown in Figure III has been closed. Later, closure was completed (Figure V).

Although every endeavour should be made to close the peritoneum underneath an implant, this is not essential. For example, there was no indication whatever of any deleterious effects on the bowel in a case in which it was necessary to leave an area measuring 6 in. long and up to 5 in. wide, over which the bowel was directly in contact with a tantalum implant (Wilson, 1956).

Wangensteen (1956) has described the use of pedicled flaps of fascia lata; but, by staging the operative repair of "bad" herniæ, such extensive operations may be avoided. In most cases of massive herniæ, the poor general condition of the patient dictates minimal operative procedures rather than one major attempt at cure.

After an incisional hernia has been reduced at operation, the abdominal wall should be examined from within, so that a small, outlying hernia is not overlooked.

If intestinal obstruction is present in a patient with an incisional hernia, the first operation should usually be limited to relief of the obstruction. Similarly, if other lesions exist in addition to an incisional hernia and require surgical treatment, they, too, should be corrected at separate stages. One exception to this is, perhaps, sterilization in the female. This restriction to one procedure at a time should apply even to patients who have both an abdominal fatty apron and an incisional hernia.

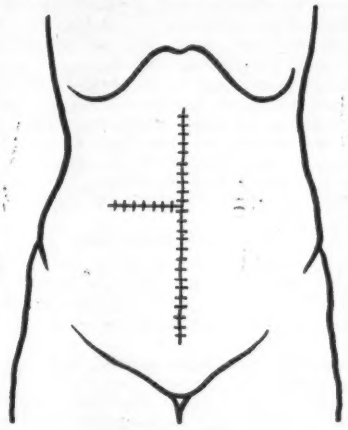

FIGURE V.
At the second stage the remainder of the hernial orifice (Figures III and IV) was closed.

The removal of such a fatty apron (lipectomy) increases the risk of infection, and should be postponed until well after the hernia has been repaired and has healed.

On the other hand, it has been suggested by Mair (1948) and others that closure of a massive hernia may be facilitated by resection of portion of the bowel.

If at operation on an incisional hernia in the upper part of the abdomen the liver is very mobile, it may be fixed in a ptosed position to give some support to the repair from within. For this purpose, the coronary ligaments may be divided to allow the liver to move downwards in the abdomen.

When an incisional hernia has followed an operation for the removal of an intraabdominal cancer, the opportunity for a "second look" for an early recurrence or for metastases should not be forgotten at the time of the hernial repair.

Post-operative Management.

The special post-operative management after operations on incisional herniæ is concerned chiefly with avoidance of tension on the suture line. For this purpose, the abdomen may be completely encircled and supported with adhesive strapping at the conclusion of the operation. This procedure is of more use in thin patients than in fat ones, for in the latter the edges of the strapping tend to roll together.

Any post-operative abdominal distension is treated as necessary by suction through an intragastric tube, by catheterization, or by the passage of a rectal tube.

There should be no straining on defæcation, and the patient should roll onto the side before sitting up.

Early ambulation should be resisted after repair of a massive hernia, the patient being kept in bed for at least two weeks. In exceptional cases this time is prolonged up to six weeks, that being the time during which sutured wounds increase in strength. Graduated exercises are commenced under supervision immediately after operation. Otherwise, especially in the very elderly, hypostatic pneumonia may ensue and embarrass the processes of

wound healing. In addition, penicillin or other antibiotic is given as a prophylactic measure against respiratory tract infections.

When ambulatory, the patient is advised to wear a well-fitting belt or corset for the next six weeks. This is applied before rising in the morning and is removed only after lying down in bed at night. If at all possible, such patients should do no heavy work thereafter, and they should be especially advised against pushing a motor-car, lifting children and similar heavy domestic activities.

As a routine measure, the exhibition of one of the tranquillizers is useful in the post-operative period. Then, the patient who realizes the desirability of giving up smoking finds less difficulty in doing so, nausea and vomiting are reduced, and the patient becomes more willing to accept a long stay in bed.

A follow-up investigation of patients who have had operations on incisional herniæ should be maintained in a similar manner to that after operations for cancer. Otherwise, the recurrence rate may appear to be satisfyingly low, or the opportunity to repair a recurrence whilst it is still small may be missed. In the majority of cases future pregnancies should be avoided.

Sutures and Implants.

In most papers on the treatment of herniæ the emphasis is on the superiority of one type of suture material or implant over others; but, with certain exceptions, success depends more on the manner of use than on the type of material.

If the ideal suture material existed, it would possess the following qualities: the knots would remain firm; it would be inert, easily handled, easily sterilized, readily available, strong, elastic, cheap, not radio-opaque, and not palpable by the patient; and the ends would not unravel. My personal preference is for cotton sutures, for these come close to being ideal; but, undoubtedly, comparable results are obtainable with some other non-absorbable materials. Sutures of silk and silkworm gut are two exceptions to this, for they both contain foreign proteins; and, indeed, it is because of the trouble with silk that all non-absorbable sutures are often suspect.

Similarly, the ideal material for an implant would be found to possess the following qualities: it would stimulate the formation of fibrous tissue; it would not predispose to infection; it would be easily sterilized, strong, easily handled, readily available in sufficient size, smooth, elastic, not radio-opaque and not palpable by the patient; any exposed portion would become covered by second-intention healing; it would not cause adhesions if in direct contact with the bowel; and there would be no difficulty with a subsequent operation through it.

For an implant that will act as a scaffold to support the ingrowth of fibrous tissue, my preference is for tantalum mesh. This material was made popular by Koontz (1948), but it is not quite ideal, and comparable results are obtainable with certain other materials.

Summary.

Staging of the operative repair of massive incisional herniæ is recommended. By this method a series of 12 massive incisional herniæ, which would have been incurable by routine methods, have been dealt with successfully.

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A REMARKABLE OUTBREAK OF "Q" FEVER.

By R. ST. C. DWYER, J. I. TONGE AND T. R. HOFFMAN,
Department of Health and Home Affairs, Brisbane,
AND
W. SCOTT, E. H. DERRICK AND J. H. POPE,
Queensland Institute of Medical Research, Brisbane.

BETWEEN October and December, 1958, an outbreak of "Q" fever with unusual features occurred on a pineapple farm near Palmwoods, Queensland, 60 miles north of Brisbane. All six living on the farm—father, mother and four sons—in turn developed the disease. Some details of the cases are shown in Table I.

All six were admitted to hospital with a temperature that ranged from 102° to 104° F. and with headache as the prominent symptom. Physical signs, apart from fever, were not significant. There was no rash. The temperature fell to normal from one to five days after treatment was instituted with chloramphenicol.

After discharge from hospital the three youngest recovered quickly, Mr. and Mrs. X. more slowly over several weeks. Son A.'s fever recurred after seven days,

TABLE I.
Details of Cases.

Patient.	Sex.	Age. (Years.)	Onset.	Admission to Hospital.	Complement Fixation Test, *C. burneti.*	
					Date.	Reciprocal Titre.
Son B ..	M.	16	17.10.58	19.10.58	16. 2.59	64
Son A ..	M.	17	25.10.58	30.10.58	3.11.58	Negative result
				9.11.58	6. 1.59	128
Mr. X ..	M.	48	25.10.58	30.10.58	2. 2.59	32
Son C ..	M.	15	1.11.58	3.11.58	12.11.58	Negative result
					11. 2.59	32
Son D ..	M.	13	10.12.58	11.12.58	11. 2.59	32
Mrs. X ..	F.	42	19.12.58	20.12.58	2. 2.59	64

and he was readmitted to hospital with a temperature of 103° F., which dropped within a few hours, and an occasional loose cough.

The diagnosis was established in each case by complement fixation (Table I). Paired sera from two patients showed a substantial rise of antibodies against *Coxiella burneti.* Single convalescent sera from the other four had antibodies in significant titre. With all six, serological tests gave negative results for leptospirosis, typhus, typhoid, paratyphoid, brucellosis and psittacosis.

Two cases of "Q" fever in one family have been rare in Queensland; six cases are phenomenal. The circumstances leading to infection called for investigation.

The other recorded cases of "Q" fever arising in recent years within five miles of Palmwoods have been as follows: (i) July, 1955, in a dairy-farmer at Woombye; (ii) September, 1957, in a dairy-farmer at Woombye; (iii) January, 1959, in a school-girl, aged 14 years, who milked cows and goats on her father's farm at Eudlo. In these three cases, infection may be explained by the close contact with cows, and their occurrence is evidence that *C. burneti* is enzootic in the cattle of the area.

The farm of Mr. X. was visited in February, 1959. One of many such in the district, it consisted of 20 acres of hilly, well-drained country. It was well kept. The main crop was pineapples; there were also some small crops, bananas, citrus, mango and nut trees. Some areas, including the surroundings of the house, were grassed. The highest and steepest part was still wooded. There was a patch of rain forest around a soak 50 yards west of the

house, and a water-hole surrounded by bananas in a small gully 150 yards north-east of the house. The domestic water supply was from rain-water tanks supplemented by a well. An earth closet was used, with burial on the farm.

Mr. and Mrs. X had occupied the farm for about 19 years; the sons had always lived there. From September, 1958, until the end of November, Mr. X. and his sons planted pineapples. Fertilizers used included "meatworks fertilizer". There was also much activity at the time around the water-hole, as the season was dry, particularly during October (Table II). The hole was enlarged and deepened and water was obtained from it to water the pea crop. Mr. X. cannot recall leaving the farm in the month before his illness. The boys occasionally visited the cinema and a swimming-hole in a nearby creek. The three youngest attended school daily, and worked about the place during the week-ends. Mrs. X. went at times into the farm. She had, for instance, picked peas in the area near the water-hole. She also washed the dirty work-clothes.

There were one dog and one cat, but no cows, sheep or goats. Fowls were kept. One neighbour owned a cow. A vendor delivered fresh milk daily from a dairy a few miles away at Woombye. Bandicoots were plentiful around the soak and around the waterhole. In places the ground

TABLE II.

Rainfall at Palmwoods (in Inches).

Period.	1958	Mean of 30 Years.
August	1·89	1·84
September	2·17	2·29
October	0·95	3·07
November ..	3·72	4·53
December	12·69	6·77
Whole year ..	87·27	65·16

was dotted with their excavations. Rats were occasionally caught by the cat. Possums, wallabies and echidnas were present in the locality. Cane toads (presumably *Bufo marinus*) were occasionally seen in the well. No birds nested in the ceiling. Early in the period fleas were numerous, presumably from dog and cat. Ticks were prevalent in the grass and weeds from September to November, and occasionally were found on the dog. March flies and mosquitoes were present at times.

Infection had obviously arisen on the farm—the common locale of all the patients—and the infective focus had persisted for some time, as two months elapsed between first and last cases; but the source and mode of infection were obscure. There had been no contact with cattle or sheep, which has been almost invariable in Queensland cases of "Q" fever.

Case-to-case infection could not explain the outbreak. It could not, of course, explain the first case (son B.). Infection from B. cannot be invoked in the next three cases; these patients became infected during B.'s incubation period or before the fifth day of his illness (if a minimum incubation period of 11 days is accepted), and there is no evidence that "Q" fever patients are potentially infective during the incubation period or early in the illness. This leaves only the last two patients, who were at the time of infection, in contact with convalescents. Although the urine during convalescence may contain rickettsiæ, no case of transmission from this source is known to us. The type of patient reported (rarely) to have infected others has usually been one with severe pneumonitis, presumably with rickettsia-laden sputum. None of the present series had pulmonary symptoms; none called for X-ray examination.

As the family drank raw milk, attention was directed to the dairy herd from which this came. Neither dairy-farmer nor milk-vendor had been ill. No fever cases were reported among the other 80 customers. There were 43 cows in the herd. One had calved in September and one in November. In February and April, 1959, blood was taken from 42 cows and tested by complement fixation; two reacted at a titre of 1:8, which is not regarded as indicating infection; the rest did not react. In March, milk was collected from 37 cows and divided into six pools, and 5 ml. of each pool were inoculated into each of two guinea-pigs. One of each pair was sacrificed at seven days, and liver-spleen was subinoculated into another pair. After five to six weeks, sera of the three remaining guinea-pigs from each pool were tested for complement-fixing antibodies to *C. burneti*; all gave negative results.

The "meatworks fertilizer" aroused interest, as the meat-works where it is prepared is a centre of "Q" fever infection. All the males had handled it, and it was abundant on the clothes washed by Mrs. X. Inquiries disclosed that in its preparation bones, gut, offal and carcases were steamed under 50 pounds' pressure for six hours at 300° F.; fats and oils were taken off and the remainder was dried, ground into powder and bagged. Such treatment would be expected to destroy *C. burneti*. Moreover, this fertilizer had been used on the farm for ten years and was in general use in the district. In February, 1959, a sample was collected from the farm, and 1 ml. of a 20% suspension was inoculated into each of eight guinea-pigs; none developed antibodies to *C. burneti*.

The possibility of an animal reservoir of *C. burneti* on the farm was next investigated. Twenty bandicoots, *Isoodon macrourus* (Gould), one possum, *Trichosurus vulpecula* (Kerr), and five rats, *Rattus rattus* (Linné), were trapped between April 15 and October 28, 1959. A saline suspension was prepared from a pool of liver, spleen, kidney, lung, heart and brain; 0·5 ml. was inoculated intraperitoneally into each of six weaned mice, and (except with the rats) 2 ml. were inoculated into each of two guinea-pigs. Three seven-day and three fourteen-day intraperitoneal passages were made in the mice, and Giemsa-stained smears of enlarged spleens were examined for rickettsiæ. Subinoculation and serological testing of guinea-pigs were carried out as with the milk samples. Three bandicoots were thus found to be infected with *C. burneti*, titres of from 1:64 to 1:512 being found in the sera of inoculated guinea-pigs. These three were captured on August 4 and 27 (near the soak), and on October 6 (near the waterhole). With one of the three, infection was confirmed by the demonstration of rickettsiæ in the spleens of inoculated mice.

None of the mice or guinea-pigs inoculated from the possum and rats became infected. The sera of the bandicoots, possum and rats showed no agglutination against *C. burneti*. In the complement-fixation test, one bandicoot gave a titre of 1:8, four were anti-complementary, and the rest gave negative findings. The possum's serum reacted positively at 1:16, suggesting previous infection. The rat sera gave negative results.

There is earlier evidence that *C. burneti* is enzootic in bandicoots in the Nambour-Palmwoods area. Records of the Laboratory of Microbiology and Pathology show that in 1937 and 1938 *C. burneti* was isolated by guinea-pig inoculation from one of 55 bandicoots captured there; the serum of three of 67 bandicoots contained agglutinating antibodies. The present study indicates that there was an active focus of infection among the bandicoot population on the farm in 1959, and this appears to have been the likely source of the human infections in 1958.

Ticks in various stages were collected from the bandicoots as follows (only one was tick-free):

Ixodes holocyclus Neumann	160
Ixodes tasmani Neumann	3
Hæmaphysalis humerosa Warburton and Nuttall	131
Hæmaphysalis bancrofti Warburton and Nuttall	2

After identification, they were stored at −70° C. until inoculated into mice and guinea-pigs. For this they were divided into five pools, washed with saline and then ground and suspended in saline. No ticks were found on the possum. Two specimens of *H. humerosa* from one *R. rattus*

were also inoculated. *C. burneti* was not isolated from the ticks.

On the assumption that bandicoots were the source of the outbreak (there seems no alternative), the question arises as to the mode of transfer from bandicoot to man. The prevalence of ticks at the relevant time suggests that they may have been the factor. However, supporting evidence for this is lacking. Mr. X. does not remember being bitten by a tick, and Mrs. X., although she noticed ticks on the men's clothes, is sure she was not bitten by one. Ticks become prevalent every year in the spring.

Inhalation is the common method of entry of *C. burneti*, and the means for it were at hand. During the dry periods, dust was readily stirred up from the ground, and particularly around the waterhole, it might have become contaminated from the bandicoots or their ticks. Another possibility was contact with the water and mud of the waterhole. All the males took part in enlarging the hole and became covered with mud and water. Mrs. X. was exposed to dust and mud when she handled the work clothes.

A puzzling question is why there was such an intense infectivity on one fruit farm only, and in 1958 only. *C. burneti* has been enzootic in bandicoots in the district for years. In general, the affected farm adopted similar methods to those of the many fruit farms around. The dry spring of 1958 prevailed throughout the district. The X. family had lived for 13 to 19 years on their farm, and had carried out much the same annual routine prior to 1958, but without infection. In this respect the special activity in and around the waterhole appears significant, for it would not have been paralleled on other farms and in previous years. In some way it may have brought man and rickettsia into intimate contact.

Summary.

Between October and December, 1958, all six people living on a pineapple farm—father, mother and four sons—developed "Q" fever.

The usual sources of infection—cattle and sheep—were absent; but a potential source was found in bandicoots (*Isoodon macrourus*). Three of 20 captured on the farm carried *C. burneti*.

Infection may have arisen from inhalation of dust or droplets of water contaminated by bandicoots. There was a suggestive association with activity in and around a waterhole.

Acknowledgements.

We are indebted to Dr. J. E. Mackay, Dr. J. E. Trotter, Dr. A. S. Raine and Dr. J. C. Moffatt of Nambour for information and sera from the patients, to Mr. E. F. Gusthart, Stock Inspector, Nambour, for blood and milk from the dairy herd, to Dr. F. H. S. Roberts and Mr. R. Domrow for identification of ticks, and to the Brisbane Weather Bureau for rainfall records.

A PHYSIOLOGICAL BACKGROUND TO SPASMODIC DYSMENORRHŒA.[1]

By WILLIAM J. GARRETT, M.B., D.Phil., F.R.C.S. (Ed.), M.R.C.O.G.,

Queen Elizabeth II Research Institute, University of Sydney, and the Royal Hospital for Women, Paddington.

PRIMARY, intrinsic or spasmodic dysmenorrhœa is a condition characterized by colicky lower abdominal pain during menstruation. The pain commonly begins in later adolescence and not infrequently persists with each menstrual period until the first pregnancy. Spasmodic dysmenorrhœa rarely appears in the first twelve months after the menarche. It is widely believed that the pain is psychological, but this explanation does not fit all the facts. In any group of young women it would not be surprising to find considerable psychological overlay with any recurrent painful condition. How much more must this be so when the condition involves the genital organs! The over-anxious mother, the apprehension and fear of a pain which is certain to come back each month and the interference of this pain with the victim's social engagements may so dominate her every thought that the actual underlying cause of the pain may be overlooked. From histological studies of the endometrium, spasmodic dysmenorrhœa has been attributed to an imbalance in the normal œstrogen-progesterone equilibrium; but this work has not been supported by assays of these hormones in the blood and urine in dysmenorrhœic patients. Uterine hypoplasia has figured in the list of causes; but in considering hypoplasia as an ætiological factor, it must be remembered that many patients with dysmenorrhœa have normal uteri and many patients with hypoplastic uteri do not complain of dysmenorrhœa. More likely is it that spasmodic dysmenorrhœa is more common with hypoplastic uterus. An association may be established, but not ætiology.

Dysmenorrhœa is essentially pain, and whatever other complaints the patient may have, it is pain which is predominant. Spasmodic dysmenorrhœa reaches its height on the first or second day of the menstrual period, and is therefore to be distinguished from premenstrual tension, a condition characterized by an increasing feeling of malaise, pelvic discomfort, pain in the breasts and general irritability in the week immediately preceding menstruation. Premenstrual tension is usually relieved by the onset of the menses.

A Physiological Explanation.

The contractility of the normal uterus has been studied by placing a small water-filled balloon in the lumen of the uterus to allow the recording of pressure changes in the uterus with a tambour or mercury manometer on a kymograph. This methods reveals two distinct patterns of uterine contractility in the course of the normal menstrual cycle. The first or basic pattern is of rapid, feeble contractions or *A* waves which are present throughout the cycle. After ovulation a second type of contractions or *B* waves appears. They are superimposed on the *A*-wave activity and increase in intensity and duration until the onset of menstruation. *B* waves, having reached their maximum on the first day of the period, decrease as the period continues and finally disappear with the end of menstruation. Pressure recordings show that *A* waves are very small and develop pressures of only 0·2 to 1·0 mm. of mercury at intervals of 10 to 50 seconds. Their frequency is greatest at the time of ovulation. *B* waves, in sharp contrast, are much more powerful, and in the last few days of the cycle develop pressures of 20 to 60 mm. of mercury at intervals of 2 to 8 minutes (Figure I, A, B). On rare occasions contractions of up to 100 mm. of mercury are noted (Moir, 1934, 1944; Henry and Browne, 1943; Garrett, 1956).

In patients with spasmodic dysmenorrhœa, Moir (1936) has found that the slow, strong *B* waves of the second half of the menstrual cycle develop unusually high pressures during an attack of dysmenorrhœa. With intrauterine balloon recordings, he has recorded pressures of 120 mm. of mercury or more in such cases as compared with the normal 20 to 60 mm. of mercury. Furthermore, he has been able to show that these powerful contractions coincide in time with the actual cramping pain in an attack of dysmenorrhœa. While recording uterine activity, Moir gave the patient a sphygmomanometer bulb to hold and instructed her to squeeze it whenever she felt the pain. He connected the tubing from the bulb to a tambour and so was then able to record the patient's subjective sense of pain on the same kymograph as her uterine contractility. He showed that the onset of pain followed the onset of the uterine contraction after a short delay, and that the pain was always present when the intrauterine pressure exceeded 80 to 100 mm. of mercury (Figure I, C).

[1] From the British Medical Association Prize Essay for 1959, "Disordered Uterine Action".

Women experiencing spasmodic dysmenorrhœa may have occasional pain-free menses, and when this occurs, the endometrium is found to be in a non-secretory phase. The cycle has then been judged anovulatory (Novak, 1952). Recently it has been shown that in anovulatory cycles the large, powerful *B* waves which Moir associated with the pain fail to appear in the second half of the cycle (Garrett, 1959). Further support is hereby provided for the belief that the pain of each spasm of dysmenorrhœa is associated with individual contractions of the uterus. When the intrauterine pressure rises above 80 mm. of mercury, circulation in the uterine wall must be seriously interfered with, as the uterine arterial blood pressure is equalled if not exceeded. Obviously this state of affairs must be much

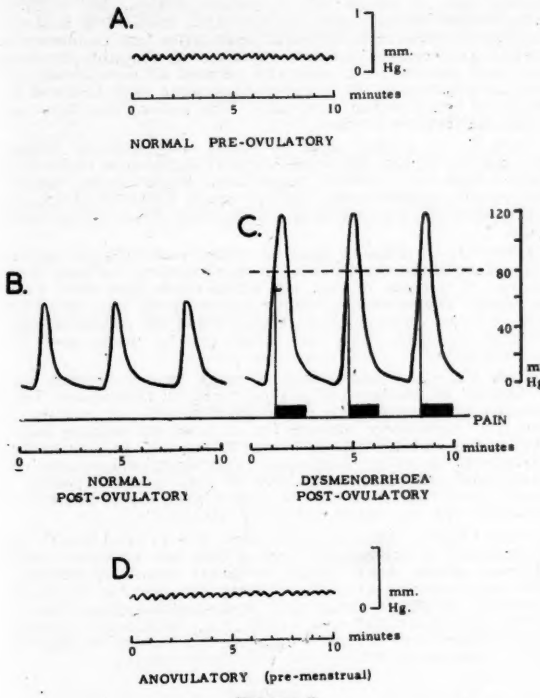

FIGURE I.
Schematic representation of contractile waves recorded from the human uterus *in situ*.

intensified in those cases in which pressures of 100 to 120 mm. of mercury have been recorded from the uterine cavity during contraction, and under such conditions a period of ischæmia and probably complete anoxia must follow. It is not difficult therefore to understand how this mechanism in the uterus will produce pain, when one considers the nature of cardiac pain in coronary insufficiency, in which there is a direct relationship between exercise, oxygen demands of the myocardium and the subjective sense of pain in the patient. A similar comparison could be drawn from the pain and tenderness in skeletal muscles after prolonged and unusual exercise.

Possibilities of Treatment.

Considering the mechanism underlying the pain of spasmodic dysmenorrhœa, we have a working basis for any attempt at rational treatment. The standard method of dilatation of the cervix under anæsthesia hitherto used brings relief for the succeeding two or three menstrual periods, and in a number of cases for longer. It could be suggested that in nulliparous women the cervical canal is very narrow; this, with the advent of unduly powerful uterine contractions, may have a relative valve-like action, especially in the presence of blood clot. The pressure in the uterus would then rise rapidly and reproduce the picture described by Moir. Dilatation of the cervix must release this mechanism and so relieve the pain. If any new surgical method is to be designed to cure spasmodic dysmenorrhœa once and for all by an operation on the uterus, it will need to enlarge the cervical canal permanently, and there can be many objections to this. We are left, therefore, with the unsatisfactory present procedure of simple dilatation.

Attempts at curing the pain of spasmodic dysmenorrhœa with drugs have met with a singular lack of success. From the mechanism described in this paper, it is obvious that a drug would need to reduce the strength of the uterine contractions so that the embarrassingly high intrauterine pressures were never reached. There have been no publications on this subject; but from references to a similar state of affairs in the pregnant uterus in labour, it is probable that the only drugs which reduce the strength of uterine contractions adequately are chloroform, ether and halothane ("Fluothane") (Embrey, 1940; Embrey, Garrett and Pryer, 1958), all of which are general anæsthetics and therefore of no use for a recurrent pain. There is little reason to believe that the third ovarian hormone, relaxin, has any useful action. Early clinical reports suggested that relaxin inhibited uterine contractility, and there was some support for this by objective recording methods (Posse and Kelly, 1956). However, more recent studies cast doubt on these earlier findings, and the consensus of opinion is that any effect in the human uterus is negligible (Kelly and Posse, 1956; Embrey and Garrett, 1959).

So-called spasmolytic drugs have been recommended in the treatment of spasmodic dysmenorrhœa; but their action, as far as the uterus is concerned, is based on false premises. These drugs block the peripheral ganglia of the parasympathetic nervous system and reduce motility in the gut. Parasympathetic nerves have never been found in the uterus, and it has yet to be shown that the uterus is under the control of the parasympathetic nervous system in any way (Embrey, 1958). It is highly unlikely, then, that these drugs will have a useful effect, and this is borne out in practice. What direction the myometrium has from the autonomic nervous system is most probably mediated through the sympathetic nerves as a balance between adrenaline and noradrenaline, both of which are released at sympathetic nerve endings (Mann and West, 1951). Adrenaline inhibits the large contractions of menstruation and noradrenaline stimulates them (Garrett, 1955), so that any drug modifying this balance in dysmenorrhœa will have to block the local effect of noradrenaline without interfering with the action of adrenaline. To date, no such drug is available, so the immediate outlook for drugs to relieve spasm of dysmenorrhœa is poor.

One other approach remains. The uterine pain receptors transmit their impulses through sympathetic fibres to the central nervous system, and to interference with this pathway the operation of pre-sacral neurectomy owes its success. The operation, although not technically difficult, carries with it the surgical risk of any laparotomy, so by its very nature is best reserved for the more crippling cases of dysmenorrhœa. Presacral neurectomy is satisfactory, in that it relieves the pain of dysmenorrhœa, but it has the theoretical disadvantage that it is a treatment of the effect rather than the cause.

Summary.

1. Each spasm of pain in dysmenorrhœa is related to an exceptionally powerful uterine contraction. Such contractions most probably lead to a state of ischæmia in the uterine wall and hence to pain.

2. If attempts at the treatment of spasmodic dysmenorrhœa are to be wholly successful, the method or drug used must prevent the raising of intrauterine pressure either by providing adequate drainage of the uterine cavity or by reducing the strength of the myometrial contractions. No treatment so far devised achieves this aim.

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THE BURNING SKIN SYNDROME: TREATMENT WITH HYDROXYCHLOROQUINE.

By R. F. BUTTERWORTH, M.D.,

Mount Royal Geriatric Unit, Parkville, Victoria.

PERSISTENT burning sensations of the skin are met with after trauma, associated with rheumatoid arthritis, and in the feet of elderly persons otherwise healthy. The legs, forearms and face are most frequently affected. Discomfort is usually worse at night, and may be so great that the warmth of the bedclothes becomes unbearable and the patient will lie exposed to the cold to gain some relief. No neurological abnormalities can be detected (*Lancet*, 1958).

Whatever the underlying cause, distress is real enough. The unprompted description — the limb "feels on fire" — is used with curious consistency by even the most literal-minded patients. There is nothing to suggest a neurotic basis for the complaint. In all the cases described below the patients appeared to be emotionally mature and faced their disabilities with courage and resolution.

No effective treatment has been hitherto described. Claims made for the vitamin B complex have been disproved (Bibile, 1957). The chance remark of a patient (Case I) on the effects of "Mepacrine" led to a trial of hydroxychloroquine sulphate in the form of "Plaquenil" (Winthrop Laboratories), a drug known to be less toxic, but to have similar properties. Eight cases in which it appeared to be effective are described.

CASE I.—The patient was a woman, aged 52 years, with severe rheumatoid arthritis of six years' duration, who complained of intense burning feelings in the hands, forearms and face. Œstrogens, steroids and tranquillizers had all been tried over long periods without result. The patient herself remarked that five years previously a course of "Mepacrine", whilst having no effect on the joints, had abolished the burning for a long period. Hydroxychloroquine was given, no special comment being made. Within two days the patient volunteered that she felt cooler, and within a week the burning had completely disappeared. A year later there had been no return of the complaint.

CASE II.—A woman, aged 45 years, had a ten years' history of severe rheumatoid arthritis, and had been confined to a chair for the last three years. Intermittent severe burning feelings of the arms and face had been present for the past two years; œstrogens and steroids produced no improvement. An inert tablet identical in appearance with hydroxychloroquine was given for one week without effect. When the active drug was substituted, unknown to the patient, there was complete relief within five days.

CASE III.—A woman, aged 37 years, had for nine years suffered from rheumatoid arthritis, and for two years from burning of all limbs. Steroids and blood transfusions improved her general condition, but did not affect the burning syndrome. The latter was abolished by hydroxychloroquine within one week.

CASE IV.—A retired policeman, aged 69 years, was in good health, and of robust and optimistic nature, but suffered from severe burning pain in the right foot. This followed removal of the right femoral head after an unsuccessful acrylic arthroplasty for osteoarthritis of the hip. Burning had been present for a year and resisted all local treatment. The administration of hydroxychloroquine was followed by complete relief within four days. The patient has remained symptom-free for a year.

CASE V.—A woman, aged 35 years, complained of intense burning of the legs following bilateral Girdlestone operations, twelve and four months previously. Barbiturates, steroids and placebo tablets were without avail. Hydroxychloroquine administration was followed by complete relief within eight days.

CASE VI.—A woman, aged 73 years, was in good health; no abnormality was found on examination, but she complained of burning of the feet, worse when they were warm at night. The symptom had been present for one year. The use of hydroxychloroquine was followed by complete relief within ten days. She was symptom-free when seen two months later.

CASE VII.—A woman, aged 67 years, was a diabetic with a history of moderately severe cerebral thrombosis three months previously. She complained bitterly of burning of the feet, apparently present for at least six months before the stroke and unaffected by it. There was no evidence of peripheral neuritis. Response to hydroxychloroquine was rapid, and the patient was free of this symptom within a week. She remained untroubled during two months in hospital, but has since been lost sight of.

CASE VIII.—A man, aged 80 years, was in good health, but complained of intense burning of the feet especially when he was sitting down in the evenings watching television. The circulation in his legs appeared normal. Placebo tablets identical in appearance with hydroxychloroquine had no effect; but when the active drug was substituted, improvement commenced within 48 hours. No further trouble has been experienced during the past four months.

Comment.

In the cases described — three in association with severe rheumatoid arthritis, two following severe trauma to hip joints, and three the "burning feet" syndrome of elderly people — the patients were all completely relieved by hydroxychloroquine sulphate. The mechanism of action is not clear, but relief appears to persist for long periods. In all cases the dosage employed was 600 mg. per day, divided into three equal doses. This was given for one month except in the rheumatoid cases, when it was given for three months as part of the general treatment.

Hydroxychloroquine is the least toxic of the antimalarial drugs, and has been widely used in the treatment of rheumatoid arthritis and lupus erythematosus (Kersley and Palin, 1959; Mullins *et alii*, 1956). Its "anti-burning" effect does not, however, appear to have been previously noted. Serious complications are few, though misty vision due to corneal deposition is well known (Sorsby, 1959).

It is fully realized that the foregoing is no proof of the efficacy of the drug, and by modern standards is quite inadequate as a therapeutic test. However, the results are sufficiently striking to justify a full-scale double-blind test on the lines of that carried out so elegantly by Bibile (1957).

The possibility that a combination of hydroxychloroquine and œstrogen in menopausal flushing may allow the amount of the latter to be decreased without loss of effect is being investigated.

Summary.

1. The burning skin syndrome is discussed, together with the very real distress caused thereby.

2. The absence of effective remedies is noted, and the reason is given for trying hydroxychloroquine.

3. Eight consecutive cases are described, in which the condition was rapidly and completely relieved by hydroxychloroquine.

4. Comments are made on the dosage employed and the relatively low toxicity of the drug.

5. The unreliability of this small series as a proof of the drug's efficacy is strongly emphasized; but the claim is made that results are sufficient to justify a full-scale "double-blind" trial.

6. The possibility is suggested that a combination of œstrogens and hydroxychloroquine may be useful in menopausal flushing.

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Reports of Cases.

MEGALOBLASTIC ANÆMIA ASSOCIATED WITH ANTICONVULSANT DRUG THERAPY.

By BERTA UNGAR, M.D.,

AND

D. C. COWLING, M.D., M.R.A.C.P., M.C.P.A.,

Department of Clinical Pathology, Royal Melbourne Hospital.

MEGALOBLASTIC ANÆMIA associated with anticonvulsant drug therapy was recently the subject of a review by Stokes and Fortune (1958), who analysed the reported findings in 36 cases and presented a further case of their own. To this collection Wilkinson (1959) has added another Australian case. Stokes and Fortune (1958) suggest that the anticonvulsant drugs have an antifolic acid action, which is too mild to produce megaloblastic anæmia in the majority of epileptics unless one or more co-factors operate. Although the reported incidence of megaloblastic anæmia in patients receiving anticonvulsant therapy is very low, we suspect that the actual incidence is probably higher. During the last four years we have seen three other cases of megaloblastic anæmia in patients receiving anticonvulsant drug therapy; they all responded to folic acid therapy, but the cases were not remarkable in any other way. The case presented in this paper emphasizes the importance of not accepting folic acid deficiency as the sole explanation of megaloblastic changes in patients receiving anticonvulsant drug therapy.

Clinical Record.

Mrs. A., aged 48 years, was admitted to the Royal Melbourne Hospital on August 24, 1959, because she had developed severe anæmia. She had suffered from epilepsy since the age of 11 years, had had chorea at the age of 14 years, and had been a voluntary boarder at a mental hospital since 1954. She had had about one seizure per month until January, 1957, when she was put on to "Dilantin" (100 mg. per day) and phenobarbitone (16 mg. per day). During 1958 she had no further seizures. The predominant features of her mental state had been an hysterical personality, hypochondriasis and at times suicidal depression, which had responded to electroconvulsive therapy. She had always had a poor appetite, and been underweight and constipated. During the three months before her admission to this hospital she had developed signs of progressive anæmia, with increasing anorexia, lassitude, apathy and unsteadiness. There was no history or other evidence of blood loss or malabsorption.

On physical examination, the patient appeared extremely pale and sluggish. The tongue was pale and atrophic. There were no petechiæ, no lymph node enlargement and no splenomegaly, but the liver was enlarged to 4 cm. below the right costal margin. The heart was moderately enlarged with signs of mitral stenosis and incompetence, and the jugular venous pressure was raised; but the lungs were clear and there was no peripheral œdema. Examination of the nervous system showed the following interesting abnormalities: hyperactive knee and elbow jerks, diminished ankle jerks and bilateral extensor plantar responses; a diminished position sense; marked calf tenderness and muscle wasting.

The initial clinical impression was that the patient had pernicious anæmia with subacute combined degeneration of the spinal cord and old-standing rheumatic heart disease; but she had obvious malnutrition, and it was suspected that she might have megaloblastic anæmia due to anticonvulsant drugs, although folic acid deficiency alone could not have accounted for the signs of subacute combined degeneration of the cord.

A number of investigations were undertaken. On August 25, the hæmoglobin value was 3·4 grammes per 100 ml. of blood, the erythrocytes numbered 920,000 per cubic millimetre, the colour index was 1·27 and the leucocytes numbered 6000 per cubic millimetre. The erythrocytes were normochromic, but showed marked anisocytosis with definite macrocytosis. The platelets were somewhat reduced in number in the films, and the neutrophil polymorphonuclear leucocytes showed marked nuclear hypersegmentation. Reticulocytes amounted to 1%. The packed cell volume was 10% and the erythrocyte mean corpuscular volume was 108 c.μ, with a mean corpuscular hæmoglobin value of 37$\gamma\gamma$, and a mean corpuscular hæmoglobin concentration of 34%. The bone marrow was very hypercellular, and erythropoiesis was hyperactive and distinctly megaloblastic; granulocytopoiesis was also hyperactive, and numerous giant band forms were present. A histamine test meal after a subcutaneous dose of 0·25 mg. of histamine showed a high concentration of free hydrochloric acid in the stomach (92°).

Treatment had to be commenced before other investigations could be undertaken. Progress blood and bone marrow findings will be discussed in relation to the course of the disease and treatment. Gastric biopsy performed on September 3 revealed normal gastric mucosa. Jejunal biopsy performed on September 18 revealed normal jejunal mucosa, except for the fusion of several villi at one site only. The absorption of radioactive vitamin B_{12}, measured by a modification of the urinary excretion test of Schilling (1953), was normal. The radioactivity of the urine excreted in 48 hours amounted to 20% of that of an oral dose of 1 μg. of ^{58}Cobalt-labelled vitamin B_{12}. Serum was collected for estimation of the vitamin B_{12} level before any treatment was undertaken, and was stored at −20° C. until the assay could be performed. The vitamin B_{12} concentration of the serum estimated by the microbiological assay method of Hutner *et alii* (1956), using *Euglena gracilis*, was found to be 63$\gamma\gamma$ per millilitre. This value is well below the lower limit of normal in this laboratory, and is of the order usually seen only in pernicious anæmia.

"Dilantin" therapy was discontinued forthwith, but phenobarbitone administration was continued at the rate of 90 mg. per day throughout. In view of the signs of subacute combined degeneration of the cord, it was decided to start treatment with vitamin B_{12} and liver extract,

and not with folic acid. She received "Combex" (2 ml.) and "Campolon" (5 ml.) daily by intramuscular injection throughout her stay in hospital. She was also given vitamin B_{12} (1000 μg. per day) from August 25 to September 4, and 100 μg. only on alternate days from September 5 to October 1. On August 27, there was no sign of response to vitamin B_{12} or liver therapy; the bone marrow was still megaloblastic, and the peripheral blood picture remained unchanged. She was therefore given folic acid in a dosage of 20 mg. daily by mouth, starting after the bone marrow biopsy on August 27 and continuing throughout her stay in hospital. On August 31 she showed a reticulocyte response of 30%, presumably to folic acid administration, and on September 1 the bone marrow was normoblastic, although occasional giant band forms were still present. On September 7 the bone marrow was normoblastic and normal. The hæmoglobin value had risen to 5·4 grammes per 100 ml. of blood, the erythrocytes numbered 1,600,000 per cubic millimetre, 8% being reticulocytes, and the blood film showed anisocytosis, macrocytosis and polychromasia of the erythrocytes; a small proportion of the erythrocytes were slightly hypochromic in appearance. She was therefore given ferrous sulphate (0·6 gramme by mouth per day) starting on September 9, and "Ferrivenin" (100 mg. intravenously per day) starting on September 14, both courses being continued throughout her stay in hospital. On September 14 her hæmoglobin value had risen to 6·9 grammes per 100 ml. of blood and her erythrocyte count to 2,200,000 per cubic millimetre, 3% being reticulocytes. On September 18 her hæmoglobin value had risen further to 8·8 grammes per 100 ml. of blood, and the proportion of reticulocytes was 6%. On September 18 and 19 she had three major epileptic seizures and she was therefore given "Mysoline" orally (750 mg. per day), starting on September 19 and continuing throughout her stay in hospital.

Her blood picture and her clinical condition continued to improve and the signs of subacute combined degeneration of the cord had largely subsided when she was discharged to a convalescent hospital on October 1. The administration of iron by mouth, phenobarbitone and "Mysoline" was continued, but all other medication was discontinued after her discharge. On review in the out-patient department on November 10 she was found to be very well. Her hæmoglobin value was 14·0 grammes per 100 ml. of blood, the erythrocytes numbered 5,000,000 per cubic mm. and the blood film showed normochromic normocytic erythrocytes with slight anisocytosis.

Discussion.

This patient is particularly interesting, in view of Stokes and Fortune's (1958) opinion, expressed as follows:

> The rarity of megaloblastic anæmia occurring in epileptics on anticonvulsant drug therapy suggests that the antifolic action of the drugs is too mild to cause the anæmia in most cases unless one or more co-factors operate at the same time.

In this patient there was folic acid deficiency combined with vitamin B_{12} deficiency and latent iron deficiency. The vitamin B_{12} deficiency was not due to pernicious anæmia, but was apparently due to an inadequate nutritional intake, or perhaps to temporary interference with vitamin B_{12} absorption from the gut by anticonvulsive drugs. The vitamin B_{12} deficiency was of such severity as to produce signs of subacute combined degeneration of the cord. Her bone marrow remained megaloblastic on vitamin B_{12} therapy alone, but became normoblastic when folic acid was given as well, thus demonstrating the existence of concomitant folic acid deficiency.

Caution dictated the necessity of starting treatment with vitamin B_{12} alone, and not with folic acid alone, to gauge the individual response in this case, as it was feared that folic acid alone might have caused an exacerbation of the subacute combined degeneration of the cord. Vitamin B_{12} and folic acid could have been given together initially; but this course would have prevented the demonstration of folic-acid deficiency in the presence of vitamin B_{12} deficiency in this case.

Conclusion.

Severe vitamin B_{12} deficiency can exist in the absence of pernicious anæmia and of malabsorption from the usual causes, and cannot therefore be excluded by the presence of normal gastric mucosa, normal free hydrochloric acid secretion by the stomach and normal absorption of radioactive vitamin B_{12} from the gut. The only means of assessing whether vitamin B_{12} deficiency exists or not is by the microbiological determination of the vitamin B_{12} level in the serum.

The findings we have described confirm that more than one factor operating at the same time may be responsible for the development of megaloblastic anæmia in a patient receiving anti-convulsant drug therapy. Vitamin B_{12} deficiency cannot be excluded in such cases unless the serum vitamin B_{12} level is found to be normal.

Megaloblastic anæmia due to vitamin B_{12} deficiency does respond to folic-acid therapy. Hence the megaloblastic changes reported in the literature in association with anticonvulsant drug therapy cannot safely be ascribed to folic-acid deficiency alone in those cases which offer no information as to the vitamin B_{12} level of the serum.

It is clearly important to keep patients who have to take anticonvulsive drugs on a well-balanced diet made sufficiently attractive to be edible. It is not recommended that patients be given folic-acid tablets as a routine in combination with anticonvulsant drugs, as this may mask concomitant vitamin B_{12} deficiency and precipitate subacute combined degeneration of the cord.

Acknowledgement.

We wish to thank Dr. J. L. Frew, Honorary Physician to In-patients, Royal Melbourne Hospital, for permission to study and report this case.

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Reviews.

The Practice of Clinical Child Psychology. By A. O. Ross, Ph.D.; 1959. New York, London: Grune and Stratton Limited. 9" × 5¾", pp. 286, with illustrations. Price not stated.

ALAN O. ROSS, Chief Psychologist to the Pittsburgh Child Guidance Centre, has in this book attempted to fill the gap between works on child development and clinical psychology. He believes that clinical child psychology is a distinct professional speciality, with its chief outlet in the child guidance clinic.

An opening chapter deals with the child guidance team, which basically consists of child psychiatrist, clinical child psychologist and psychiatric social worker. Though in some clinics the child is regarded as the patient, the author stresses that the patient is the family unit and the therapist is the whole team. It follows from this that "neither mother nor child is the more important patient and conversely neither team member has the more important task". He deplores the fact that it is often difficult to obtain fathers' cooperation in the drive for therapy. The broad scope of the problem is illustrated by the finding that 45% of children referred to the clinic have "academic difficulties", a fact which calls for close liaison with schools.

In dealing with the psychologist as a diagnostician, the author, whilst stressing the value of diagnostic tests, is careful to outline the limitations. "Our techniques do not possess the refinement necessary to evaluate parent-child reaction." In order to prevent confusion in the child's mind, the therapist should not be the person who administers the tests.

An interesting chapter is devoted to case histories of two disturbed children who had cerebral disease. The diagnosis was made largely as a result of the Bender Gestalt findings. They illustrate that "organically oriented pediatricians are often accused of ignoring emotional factors, but the harm which can be done if the psychologically oriented specialist ignores organic factors is equally great".

The author's approach to the clinical material is factual and rightly appreciative of the difficulties of classification into types. He gives case histories of children showing aggression, withdrawal, bizarre behaviour and learning behaviour. The most controversial section deals with the psychologist as a therapist. He quotes references showing that in 84·2% of 221 clinics the psychologists conduct treatment, and he refuses to be drawn into a debate on who may conduct treatment. More relevant seems to be the question of who should conduct therapy with children. The prerequisites for a therapist include adequate training under supervision and, particularly, a personality structure which makes it possible to be comfortable with children.

As might have been expected, the book deals with the interrelations of the child guidance team. It varies in different clinics. A technique is recommended in which there is minimal contact between the child's therapist and the parent.

With regard to research, the author points out that the scope for investigation is boundless. Not the least is the need for more detailed information on the types of patients who are most amenable to therapy. In this the clinical psychologist can make a valuable contribution.

This book is authoritative and well written, has a useful list of references and is well documented. It will be read with interest by any who are faced with the problems of child guidance.

Therapeutic Radiology: Rationale, Technique, Results. By William T. Moss, M.D., with a foreword by Lauren V. Ackerman, M.D.; 1959. St. Louis: The C. V. Mosby Company. Sydney: W. Ramsay (Surgical) Limited. 9¾" × 6¼", pp. 403, with 146 illustrations. Price: £6 17s. 6d.

THIS is a fine textbook on modern radiotherapy. The references at the end of each chapter show that the author has undertaken a great deal of research in the approach to treatment of malignant disease by means of radiotherapy. He covers very adequately all aspects of malignant disease that any radiotherapist may be called upon to see and possibly treat. In each chapter he precedes the clinical management of the disease with the effects of irradiation on the normal tissues. This is all too often omitted from the usual textbooks. At the end of each chapter he critically analyses the results of radiotherapy with any of the other alternative methods of treatment. He carefully assesses the value of either, and many of his comments are worthwhile. The first chapter, in which he discusses the relationship between radiotherapists, pathologists, surgeons and physicists, is extremely well written and could be read by all interested in the treatment of cancer. Perhaps one criticism is that he deals very rapidly with cancer of the breast and has extremely little to say on the hormonal treatment of the disease. He also has little to say on radioisotopes, and this is a subject which we consider could have been elaborated on somewhat more. However, in fairness to him, he does state that it is impossible to cover the whole of radiotherapy in one book, and he gives at the end of each chapter an excellent list of references for more detailed study. This is a book which can be highly recommended for all interested in the radiotherapy of cancer.

La Coletitiasi et Alia Hepatologica, Atti del Primo Symposium Internationale di Epatologia; 1959. Edizioni Chianciano. 9¾" × 6¼", pp. 638, with illustrations. Price not stated.

THE first international symposium on hepatology held in the hydropathic establishment of Chianciano in June, 1958, was devoted to cholelithiasis and other hepatological conditions. The "other" is certainly an elastic term, as many pathological studies were included, such as skin affections not connected with gallstones, and even some having nothing to do with liver ailments, such as gastro-duodenal ulcers. Whether a symposium should place emphasis on research or be mainly expository regarding present-day views is not stated; but in this particular symposium of four days' duration and embracing 60 addresses and commentaries, the stress is largely on recent clinical findings and diagnostic methods. The articles are all scholarly in the best sense of the term, and a commendable candour in ascribing priority is evidenced—a welcome change from the arrogant claims of Italians in the Mussolini régime. The phrases *"secondo la technica di"* and *"con la metodica di"* appear again and again. Special praise can be given to two admirable articles dealing with the technique and the findings in the application of X rays to the exploration of the gallbladder and biliary passages contributed by Paride Stefanini of Pisa and Luigi Turano of Rome. Other articles well worth reading are concerned with liver regeneration after subtotal hepatectomy, and the digestion and assimilation of fat using triolein treated with iodine-131. Strange to say, there is almost nothing given on the disorders following reduced absorption of the fat-soluble vitamins. An emphasis on balneotherapy is present which is not to be found in medical works published in the English language; but this can be explained perhaps by the site of the symposium (which, by the way, is called international, though all the contributors are Italians, many from Bologna). The volume is attractively printed and the proof-reading has been good, as evidenced by the very few misspellings of British names—an error all too common in books and periodicals published in France.

Surgery of the Foot. By Henri L. DuVries, M.D., foreword by Karl A. Meyer, M.D., introduction by Edward L. Compere, M.D.; 1959. St. Louis: The C. V. Mosby Company. Sydney: W. Ramsay (Surgical) Limited. 9¾" × 6¼", pp. 494, with 403 figures. Price: £6 17s. 6d.

THIS book has been written to improve the teaching on foot disabilities. Previous attempts have been made to do this, but most of them have not been sufficiently practical. Foot disabilities are very common and very important, and a book such as this should be helpful to anyone who wishes to learn how to treat foot disabilities.

The author has covered the whole range of conditions that may affect feet. We consider that he could have omitted a few of the conditions that rarely affect feet; the same comment applies when the site of the lesion in the foot does not raise any special problems.

The common foot problems are dealt with very well in most cases, and most of the accepted forms of treatment are described and discussed. The illustrations of the operations are good and very helpful.

The section on flat feet is disappointing. This common condition, which is often a problem, deserves much more detailed consideration. Another thing that could be improved is the discussion of the modification of footwear. In a book of this kind these modifications should be described in the greatest detail, so that the reader can have a clear idea what to order, and can see that the bootmaker carries out the modifications correctly.

Anyone who wishes to further his knowledge of the foot will find this book most helpful. The subject is covered well, and the book is very well illustrated. It is hoped that in any future editions the author will considerably expand the section on flat feet.

Pediatric Neurosurgery. Edited by I. J. Jackson, M.D., M.Sc., F.A.C.S., F.A.C.A., and R. K. Thompson, M.D., F.A.C.S.; 1959. Oxford: Blackwell Scientific Publications Limited. 10¾" × 6¾", pp. 580, with illustrations. Price: £6 5s. (English).

THIS volume, "delineating pediatric neurosurgery from adult neurosurgery in the miniature", has been prepared by 24 contributors from all parts of the United States, and the opinions in it "represent a cross section of the country". This contrasts with the 1954 classical textbook "Neurosurgery of Infancy and Childhood", by F. D. Ingraham and D. D. Matson, which presents the views and practices of the Boston Children's Medical Centre. This latter book is, however, designed primarily for the trained neurosurgeon, whereas "Pediatric Neurosurgery" is written for "those physicians who care for neurologically ill (pediatric) patients". The volume is accordingly divided into two parts.

The first part (93 pages) has informative chapters on diagnostic techniques, electro-diagnostic methods, pre-operative and post-operative care, anæsthesia, correlative plastic surgery and conditions resembling surgical problems.

The second part, comprising 14 chapters, deals with specific neurosurgical entities, and all the important neurosurgical problems such as congenital malformations, hydrocephalus, cerebral tumours, trauma, etc., are adequately described and discussed. Especially good is the long chapter (126 pages) on tumours, intracranial and cranial, by Lyle French. This detailed and well-illustrated account can be read with pleasure and profit both by pædiatricians and by neurosurgeons. Discussing the difficulty experienced in performing percutaneous cerebral angiography in infants and young children, French advocates reflux brachial arteriograms, and

emphasizes that adequate visualization of both the carotid and vertebral systems can be readily and regularly obtained. Especially informative, too, are the chapters "Intracranial Vascular Malformations" by Lester A. Mount, and "Surgery of Involuntary Movement Disorders", by Irving S. Cooper and G. J. Bravo. Minor defects include the statement (page 145) that the hydrocephalus associated with a meningomyelocele is due to the associated Arnold-Chiari malformation, whereas in our opinion the more important obstruction is usually a coincidental stenosis of the aqueduct. Acute subdural hæmatoma in the newborn is not discussed, and the authors apparently do not realize how much help the neurosurgeon can give the neonate with acute cerebral birth hæmorrhage. In the chapter "Trauma: Peripheral Nerves", it is stated that with the exception of birth injuries, peripheral nerve injuries in childhood differ in no essential respect from those occurring in adult life; yet only two pages are devoted to birth injuries and some 17 pages to nerve injuries in general. The volume is well illustrated and there is an extensive bibliography. This is a very welcome addition to the very meagre number of textbooks on this important subject.

Forensic Medicine: Observation and Interpretation. By A. Keith Mant, M.D. (Lond.); 1960. London: Lloyd-Luke (Medical Books) Limited. 8¾" x 5½", pp. 272, with 118 illustrations. Price: 42s. (English).

THIS is a new book by A. Keith Mant, who is a lecturer in the Department of Forensic Medicine at Guy's Hospital, London. It places particular emphasis on the findings at the scene of sudden death and the conclusions to be drawn therefrom.

The book is divided into two sections. The first part deals with sudden and violent deaths, presenting the information enabling the examiner to recognize the possibility of unnatural death. The second part confines itself to medico-legal aspects of sexual offences. Photographs and case histories have been used freely to illustrate the important details.

This book, which is of handy student size, will be found very useful by all those who are called to the scene of a dead body and by those medical practitioners concerned with the examination of cases of alleged sexual assault.

Fundamentals of Modern Allergy. Edited by Samuel J. Prigal, M.D.; sponsored by the New York Allergy Society; 1960. New York, Toronto, London: McGraw-Hill Book Company, Inc. 9" x 5¾", pp. 714, with many illustrations. Price: $18.50.

SPONSORED by the New York Allergy Society, this work has 59 contributors. All are well-known people in their own fields, and yet the editing has been so well carried out that there is little overlap or repetition in the 63 chapters and four appendices.

Except for two chapters, the whole series was originally published as articles in *The New York State Journal of Medicine*; they cover the whole field of allergic practice, and there are numerous references at the end of each chapter. The work consists of five parts, each divided into a number of chapters. Part I deals with mechanisms and causal factors in allergy, Part II with tests, procedures and techniques for diagnosis of allergic conditions, Part III with manifestations and forms of allergy, Part IV with allergy in childhood and old age, Part V with general treatment and prophylaxis in allergy.

Among the subjects dealt with in the appendices are the preparation of allergenic extracts for testing and treatment, botany and allergy and the hay-fever producing plants.

There is an excellent chapter on the pathology of allergic and collagen diseases. And that on allergy, infection and the psyche could be profitably studied by psychiatrists and others who appear to think that many allergic diseases have a purely psychological basis, which every experienced allergist knows is far from correct. The relationship of allergy to sinusitis, bronchitis and other pathological conditions of the respiratory tract is dealt with in a masterly manner, and many of the newer thoughts on how allergy may play a part in various conditions such as the collagen diseases and some kidney diseases are explored in the text.

This book is designed primarily for the general practitioner with an interest in allergic conditions and for the post-graduate student attending an allergy clinic. Also the practising allergist will find it useful in bringing his knowledge up to date, and he will find provocative articles on such topics as enzyme mechanisms in allergy, histamine liberators, climate and asthma, anæsthesia for the allergic patient, and other subjects which are frequently not dealt with as fully in many standard texts.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Modern Educational Treatment of Deafness: Report on the International Congress held at the University of Manchester 15th-23rd July, 1958", edited by Sir Alexander Ewing; 1960. Manchester: Manchester University Press. Carlton: Melbourne University Press. 9¼" x 6", pp. 72 (papers) with illustrations. Price: 64s. 9d.

"Fourth Tissue Homotransplantation Conference", edited by Otto V. St. Whitelock; Annals of the New York Academy of Sciences, Volume 87, Art. 1, 1960. New York: The Academy of Sciences. 9" x 6", pp. 607. Price: $5.00.

"Classification of Pharmaceutical Preparations: A Survey of Existing Legislation", World Health Organization; 1960. Geneva: World Health Organization. 9¼" x 6¾", pp. 56. Price: 3s. 6d.

"Numerical Properties of Functions of More than One Independent Variable", edited by Otto V. St. Whitelock; Annals of the New York Academy of Sciences, Volume 87, Art. 1, 1960. New York: The Academy of Sciences. 9" x 6", pp. 677-874. Price: $3.00.

"Arthritis and Allied Conditions: A Textbook of Rheumatology", edited by Joseph Lee Hollander, M.D.; Sixth Edition, 1960. Philadelphia: Lea & Febiger. Sydney: Angus & Robertson Ltd. 9¾" x 5¾", pp. 1306, with 417 illustrations. Price: £11.

"Fractures, Dislocations and Sprains", by Philip Wiles, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S.; 1960. London: J. & A. Churchill Ltd. 9¾" x 12¼", pp. 76 with 519 illustrations. Price: 27s. 6d. net (English).

"Symposium on Salt and Water Metabolism", edited by Alfred P. Fishman, M.D.; Circulation, Volume 21, Number 5, Part Two, Supplement, May, 1960. New York City: American Heart Association; New York Heart Association. 11" x 7¾", pp. 803-1060. Price: $2.00.

"Medicinal Chemistry", edited by Alfred Berger; Second Edition, 1960. New York, London: Interscience Publishers. 10" x 6¾", pp. 1262. Price: $37.50.

"Neoplasms of Bone and Related Conditions: Etiology, Pathogenesis, Diagnosis and Treatment", by Bradley L. Coley, M.D.; Second Edition, 1960. New York: Paul B. Hoeber, Inc., Medical Division of Harper & Brothers. 10" x 6¾", pp. 880, with 649 illustrations and 31 tables. Price: $30.00.

"The Year Book of Pathology and Clinical Pathology", edited by William B. Wartman, B.S., M.D.; 1960. Chicago: The Year Book Publishers. Melbourne: W. Ramsay (Surgical) Limited. 7¾" x 5", pp. 456 with illustrations. Price: £4 19s.

"Antibiotics and Sulphonamides in Ophthalmology", by Arnold Sorsby and Joseph Ungar; 1960. London, New York, Toronto and Melbourne: Oxford University Press. 6¼" x 4", pp. 128. Price: 20s. 9d.

"Industrial Pulmonary Diseases: A Symposium held at The Postgraduate Medical School of London 18th-20th September, 1957 and 25th-27th March, 1958", edited by E. J. King, M.A., D.Sc., F.R.I.C., and C. M. Fletcher, C.B.E., M.D. (Cantab.), F.R.C.P.; 1960. London: J. & A. Churchill Ltd. 8¾" x 5¼", pp. 284 with 98 illustrations. Price: 32s. net (English).

"The Vagina", edited by Warren R. Lang; Annals of the New York Academy of Sciences, Volume 83, Art. 2, November 18, 1959. New York: New York Academy of Sciences. 9¼" x 6", pp. 77-358, with illustrations. Price: $4.00.

"Inactivation of Viruses", edited by Ernest C. Pollard; Annals of the New York Academy of Sciences, Volume 83, Art. 4, January 13, 1960. 9" x 6", pp. 513-760, with illustrations and tables. Price: $4.00.

"The Correlation of Hinged Four-Bar Straight-Line Motion Devices by Means of the Roberts Theorem and a New Proof of the Latter", by A. E. Richard de Jonge; Annals of the New York Academy of Sciences, Volume 84, Art. 3, March 18, 1960. New York: The New York Academy of Sciences. 9" x 6", pp. 75-145, with figures and tables. Price: $2.50.

"Social and Cultural Pluralism in the Caribbean", edited by Vera Rubin; Annals of the New York Academy of Sciences, Volume 83, Art. 5, January 20, 1960. New York: The New York Academy of Sciences. 9" x 6", pp. 761-916. Price: $3.00.

"Neuropharmacology", edited by Harold A. Abramson, M.D.; Transactions of the Fifth Conference, May 27, 28, and 29, Princeton, N.J.; 1960. New York: Josiah Macy, Jr. Foundation. 9" x 6", pp. 254, with 55 figures. Price: $6.00.

The Medical Journal of Australia

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THE NEWCASTLE-UPON-TYNE PÆDIATRIC SURVEY GOES ON.

FIVE YEARS ago in these pages[1] we examined at some length the first volume of the study of 1000 families living at the time at Newcastle-upon-Tyne,[2] initiated by the late Sir James Spence and enthusiastically carried on by the team he had gathered together. This first volume set out the philosophy of the study and the results of the first year's observations. On that occasion we observed that the first volume represented a pædiatric study of the first importance and that no student of pædiatrics could afford to be without it. That opinion can be expressed with equal confidence about the second volume just to hand.[3]

At the outset it is important to point out that the authors have written this second book primarily as an aid to the family doctor, to give him an over-all picture of illness in Newcastle and to help him interpret the individual child's needs against the wider scene of health and disease in this age group in that city. The philosophy of this study has been expressed in the following way: "Certain types of questions on epidemiology or the natural history of disease, in medical organization and in medical or health education, can be answered only if an appropriate group of subjects is observed over a sufficient length of time." These subjects were 1142 infants born into 1132 families in Newcastle between May 1 and June 30, 1947, and the current publication records the health and disease of the 847 who had been observed for the first five years of life. The study will be continued until 1962, when the children will be 15 years of age; so further publications can be anticipated. It is worth recording here the causes of the wastage. In the first year, 44 children died, 127 families left the city, and 4 families left the survey. In the next four years, 5 more children died, 112 were lost by removal, and 3 further families withdrew.

Not only is the second volume an account of the illnesses suffered by the children, but it also gives valuable information about the characteristics of many of the diseases as seen in children in Newcastle, the clinical features, the complications and the types of treatment used for a number of the conditions. The background details are important. Considerable space was given to environmental factors, including housing, in the first volume, and so these items are dealt with summarily in the second volume. One interesting feature, however, has been the change in the team's evaluation of the quality of mothercraft in not a few families. In the first year of the study 80% of families were judged happy and stable. At that time many were newly established and had not had to stand the test of the stresses of family life. With time these stresses began to operate, an appreciable number of families broke up as a consequence, and the weaknesses in the personalities of the parents of others became manifest by defective management. Families were classified as satisfactory when the parents were physically and mentally stable, lived in harmony with each other, and provided a home wherein the children could find affection, security and responsible care; at the end of five years the team judged there were 379 (47%) of these. The remaining families were placed in three broad categories: those lacking parental supervision (deprivation), those lacking adequate food, clothing and sleeping arrangements (deficiency), and those with such a degree of breakdown that assistance was constantly required from neighbours or from official sources (dependence). One family in 12 was deeply involved in all three of these categories. It is significant that the incidence of all illnesses was much higher in the families in one or more of these three last-named categories. In evaluating this section of the study we cannot help wondering about the preparation and skills of those members of the team who collected the sociological data. The qualifications of the medical members of such a team would never be questioned, but with the marked advances in techniques for making sociological surveys it would have been helpful to know what training and supervision the team had had in this area of its work.

The 847 children suffered 8467 significant illnesses, of which 80% were infective illnesses dominated by respiratory infections. All illnesses are discussed under five major headings: immunizing diseases, endemic family infections, diseases of the social environment, disorders of development and respiratory diseases. In each of these categories the reader will find much to interest him. The relative infectivity of the immunizing diseases and the apparent modes of spread of the diseases caused by staphylococci, streptococci and the virus of herpes simplex through the family should find confirmation from those who have found interest in this side of the story of illnesses they see in their daily work. The conditions included in the category "diseases of the social environment" may cause some eyebrows to rise: accidents all will accept, and also retarded growth (by the third year the children of semi-skilled and unskilled fathers were already 3·3 months behind the children of professional families), but will all concede that enuresis and squint (twice as frequent in the children receiving "deficient" physical care) belong here? The chapter on convulsions (included in the section on disorders of development) stresses some important facts — the significant number of children in whom the origin was unknown, the excess of non-infective convulsions in the children of semi-skilled and unskilled workers, and the strong correlations with parental deprivation and deficiency of physical care.

A large section of the book is devoted to the respiratory diseases. Here descriptions are given of the conditions seen, the numbers of children affected and the nature of

[1] MED. J. AUST., 1955, 1: 277 (February 19).
[2] "A Thousand Families in Newcastle-upon-Tyne", by J. Spence, W. S. Walton, F. J. W. Miller and S. D. M. Court, Oxford University Press, London, 1954.
[3] "Growing up in Newcastle-upon-Tyne: A Continuing Study of Health and Illness in Young Children within their Families", by F. J. W. Miller, S. D. M. Court, W. S. Walton, and E. G. Knox; 1960. London, New York, Toronto: The Nuffield Foundation, published by the Oxford University Press. 8½" × 5½", pp. 398, with many tables and illustrations. Price: 46s. 6d.

the treatment. We observe the difference between treatment of pneumonia at home and in hospital. In hospital all but two children received both penicillin and sulphonamides; the majority of those treated at home received sulphonamides only. Although the records of home treatment were incomplete, the authors note that "in 35 attacks where the facts were recorded only a third appeared to receive an adequate dose of the drug for a sufficient time. Six children received neither antibiotics nor chemotherapy".

A number of striking facts emerge from this study, including the high incidence of illness (an average of 10 episodes per child) and the significance of the quality of family life for the outcome of many illnesses. It is clear that children cannot be protected from all the immunizing diseases, but the quality of family care can reduce the severity, duration and sequelæ of these diseases and reduce the risk of infection with some of the respiratory diseases and those caused by staphylococci and streptococci. If, therefore, a survey of this kind is to be the means of improving health and not an end in itself, then some indications, for future action, on how to prevent these "family" diseases would surely not be out of place in this report.

A question that will be asked by many readers is: how far are these findings applicable to other communities — in particular, Australian communities? There is no sure answer to this question. Many of the conditions are similar. All family doctors will confirm the spread of some diseases through families, as they will the importance of maternal care and the need often to treat the whole family, and so on. We do not know the comparative incidence of the various illnesses and diseases in Australian communities; one has the impression that the incidence of many may be less, but at best this would be a guess. We can only guess, but in Newcastle-upon-Tyne they know. Perhaps someone here will be stimulated to find out soon, and indeed there are those who are thinking along these lines. In relation to such thinking one other thought, not without relevance, kept on occurring during the reading and re-reading of parts of this study: apart from confirming many notions about illness and describing some new facets, what is the significance of a survey? Surely it should never be an end in itself, but rather the beginnings of a programme designed to improve conditions. We cannot help wondering, then, whether the action stage cannot often be undertaken on the basis of existing information, or whether a preliminary survey is always necessary.

Current Comment.

HYDRAULIC KNEE ACTIONS.

FROM time to time there appear in the literature outbursts against the inadequacies of standard artificial legs and the unhappy plight of those who must wear them. Invariably these outbursts are accompanied by descriptions of new devices for providing the amputee with a nearly-natural gait—and usually, too, the new devices have been developed by people who appear to have more theoretical knowledge than practical experience in the fitting and maintaining of artificial legs. The latest of these raises several questions; it is the description of the University of Cape Town artificial limb, by C. E. Lewer Allen,[1] Professor of Orthopædic Surgery at that university. Certain provocative statements made by Professor Allen suggest that there is need for viewing the matter of artificial legs in broad perspective.

In the first place, when arose the general insistence, not confined to Professor Allen's article, that the prime requirement of an artificial leg is to secure a natural gait? Surely the underlying purpose of an artificial leg is to render the patient as efficiently mobile as possible with the maximum of comfort and security and the minimum of bodily stress. On the one hand, if this can be achieved only by sacrificing a normal-looking gait and a normal appearance, what of it? On the other hand, is it rational to aim at a normal gait for a patient who has lost a leg? The literature abounds in graphs and diagrams analysing the gait of persons who have not lost a leg, and these are used as criteria for a similar gait in amputees; but would it not be more logical to make graphs and diagrams analysing the gait of large numbers of amputees fitted with all kinds of prostheses, and establish criteria based on these?

The next point that presents itself concerns an elementary principle relating to all mechanical devices: simplicity is efficiency. Every extra refinement in function that is demanded of an artificial leg means more parts; a multiplicity of parts requires extreme precision in making them and considerable skill in assembling and maintaining them; more parts demand more housing, more bearings, and so more weight, and to save weight all these must be made lighter, and therefore weaker. But an artificial leg must be not too heavy, yet quite rugged. Moreover, few amputees can be trained to adjust or repair complicated parts, and fewer live just around the corner from a suitable mechanic who is available at all times; yet security demands not only a leg that is safe to walk on, but also one that will give trouble-free service for long periods, and the more "gadgety" the leg the less this latter requirement is likely to be met.

Not that this means that refinements are undesirable—far from it. But in practice it is found that four patients out of five receive completely satisfactory service from a simple leg, and most revert to a simpler one even after having been fitted with the more complicated type. The fifth patient needs special attention because of some complication such as an unsatisfactory stump, local or general muscular weakness, or special working conditions. It is in these complicated cases that special legs may be necessary. In practice, too, it is the exceptional patient who refuses to accept the fact of his amputation and the limitations which it inevitably imposes, and who demands an artificial leg which will do for him everything that a normal leg would do, who gives the limb-fitter his greatest worries. Nevertheless, in only a small proportion of the difficult cases does the hydraulic knee action offer an answer.

Thirdly, it is interesting to consider Professor Allen's conclusions. The first four, based on rather doubtful premises, were responsible for the foregoing discussion. Then he goes on:

> 5. A renascence is required through which fundamental research should return to the control and direction by universities. There has been a dangerous drift of scientists and their work towards specific and commercialized research. Such a drift must ultimately result in sterility in all spheres . . . .
>
> 6. The growing dangers of selfish monopolistic overcommercialization of apparatus and medical requirements of the needy are real. The world must become conscious of this and take rational action.

Exactly what this has to do with artificial legs is not clear. If it means that only theoreticians can develop elaborate mechanisms, well and good. If it also means that the purveyors of artificial legs are deliberately foisting out-of-date equipment on the public at exorbitant prices (which it might, in view of the rest of the article), then it must be pointed out that, whatever may be the position in South Africa, artificial limb makers elsewhere are eager to provide elaborate legs with the most modern

[1] *S. Afr. med. J.*, 1960, 34: 125 (February 13).

refinements at a reasonable profit—but the refinements are necessarily costly.

Finally, to return to the University of Cape Town leg: the hydraulic unit is but another variation of the usual dash-pot, combined this time with springs, giving variable cadence and knee-resistance adjusted through a cable controlling the oil-inlet. It has had only one pilot trial; field tests are planned. If we go by the description, it might reasonably be added to the already long list of hydraulic knee actions, none of which solves all the problems of artificial legs, none of which is essential except for selected cases, and all of which are extremely costly to produce and finicky to maintain.

THE ADOLESCENT PATIENT.

WIDESPREAD interest in the adolescent and his problems is apparent from current reports in the professional literature, in addition to the plethora of material in the popular Press. Unsentimental and objective views are always welcome, and several recent publications provide a wealth of valuable data for those who work with young people. Symposia on adolescence appeared in *Pediatric Clinics of North America* in February, 1960, and in *The Practitioner* of March, 1960. The former contains an excellent series of papers by 15 prominent American pædiatricians, psychiatrists and others. These cover basic concepts of physical, emotional, social and academic functions of adolescents; presented simply and sensitively, they indicate a significant trend towards "the marriage of psychiatry and pædiatrics", or what a prominent American calls "the new pædiatrics"—an attempt to incorporate precepts of mental hygiene into medical practice. In this regard, much stress is laid upon the role which the doctor may play for the adolescent in need of someone, outside his own family, with whom to relate and identify himself. Many authors also underline the vital difference between insecure authoritarianism and real authority in a changing world.

Particular interest in the welfare of young people is apparent in the field of education. J. P. Sparks,[1] school medical officer at Rugby, gives an informative survey of his work and of the opportunities prevailing in this type of practice. Dana Farnsworth,[2] director of the health service at Harvard, makes further incisive comments on the promotion of mental health in colleges, and Grahame Blaine,[3] of the same service, describes some of the emotional problems of older adolescents. Nicolas Malleson[4] remarks upon the management of examination anxiety, and a report of the eleventh conference of the British Student Health Officers' Association[5] includes a number of papers on relevant topics. M. L. Selzer[6] in a paper previously noted in these columns (August 20), points out the relative severity of some emotional disorders in students, and suggests that they are often "underdiagnosed".

Concern about delinquent behaviour is felt at all levels of professional work, and the American Academy of Pediatrics[7] has recently published a summary of a conference, together with an excellent bibliography, on the role of the pædiatrician in the prevention of delinquency; the relationship of the pædiatrician to the individual delinquent is also examined by Julius B. Richmond.[8] Lee Forrest Hill,[9] in an excellent comment on what he terms "anticipatory guidance" in pædiatric practice, points out how much the doctor can do by informed preparation of parents for some of the normal adolescent manifestations, so that they may better understand and accept them. D. T. Maclay[10] describes the subsequent progress of boys who commit sexual misdemeanours, and indicates the value of a dispassionate and optimistic attitude, as opposed to the authoritarian severity often displayed towards youthful offenders.

Some of the more specific psychiatric literature contains helpful information and suggestions for those concerned with adolescents. Methods of therapy and interpretations of adolescent behaviour are offered by Lovick C. Miller,[11] James F. Masterson,[12] and Leo Kanner.[13] Morton Levitt and Ben Rubenstein[14] point to some aspects of acting-out behaviour as a form of communication, and they suggest the importance of interpreting such activities according to their context, wherein they may sometimes be seen as a positive effort in the direction of problem solving—and hence "a moment of hope" and not merely a device for the provocation of authority. All in all, most writers share a warm and optimistic understanding of young people as individual personalities, and a readiness to meet them half-way in their struggle toward mature identity.

[1] *Postgrad. med. J.*, 1960, 36: 309 (May).
[2] *Ment. Hyg.*, 1959, 43: 358, 568 (July, October).
[3] *Student Med.*, 1959, 7: 253 (April).
[4] *Lancet*, 1959, 1: 225 (January 31).
[5] "Student Health in British Universities and Medical Schools", 1959, British Student Health Officers' Association.
[6] *Arch. gen. Psychiat.*, 1960, 2: 131 (February).
[7] *Pediatrics*, 1959, 24: 822 (November).
[8] *Pediatrics*, 1960, 26: 126 (July).
[9] *J. Pediat.*, 1960, 56: 299 (February).
[10] *Brit. med. J.*, 1960, 1: 186 (January 16).

CERVICAL CARCINOMA.

BENT SØRENSEN, of The Radium Centre, Copenhagen,[1] has attempted with success to follow up some 800 women treated at the centre over the years 1922 to 1929. In studies of this kind a choice has to be made between adequate length of follow-up and completeness of individual records, which can be ensured in a forward study. However, such complete information will not usually be available for many years, so that there is much to be said for the use of existing records. Out of 798 patients, 59 were still alive in 1954; in 20 cases it was impossible to decide whether the patient had died with or without cervical carcinoma; for 713 patients who had died this decision could be made; 6 patients could not be traced. In other words, less than 1% of patients were untraced, and in less than 3% of patients who had died a decision could not be made on whether their carcinoma was the cause of death. Sørensen points out that it is impossible to say how patients would fare without treatment.

Some interesting findings are as follows. The mortality from diseases other than the cancer itself seemed to be about average for Denmark. The probability of late recurrences appeared to fall as time went on. Some patients were actually cured of the disease, and Sørensen believes that they would not have developed a recurrence no matter how long they had lived. The report indicates that 25% of patients survived five years, 18% survived ten years, and 12% survived 20 years. Of course there was a great difference in the survival rates when they were classified by stage. But if a Stage II patient survived say ten years, her chances then did not appear inferior to a corresponding Stage I patient. By stages (I to IV) the percentages of all deaths due to recurrences were 39%, 63%, 82% and 92% respectively. About a quarter of all deaths after five-year survival were due to cancer. In 6% of all deaths due to intercurrent disease, radiation was believed to play some part. No evidence was found that the radiation provoked other cancer or released other cancers from some controlling influence.

To sum up, this is a carefully documented and scholarly report, in which tabular and graphical methods have been skilfully used. We might end with a quotation from the author on his page 103:

> More than 25% of all deaths after the fifth year and more than 10% of all deaths after the tenth year were due to late recurrences. Late therapeutic complications may be expected in about 6 per cent. of the patients. In the future, therefore, all treated patients ought to be followed until they die.

[11] *Amer. J. Orthopsychiat.*, 1959, 29: 772 (October).
[12] *J. nerv. ment. Dis.*, 1958, 127: 511 (December).
[13] *J. Pediat.*, 1959, 55: 397 (September).
[14] *Am. J. Orthopsychiat.*, 1959, 29: 622 (July).

[1] "Late Results of Radium Therapy in Cervical Carcinoma: A Clinical-Statistical Study on 798 Patients Treated at The Radium Centre, Copenhagen, During the Period 1922-1929", by Bent Sørensen; *Acta Radiologica*, Supplement 169; 1958. Stockholm: Acta Radiologica. 9½" × 7", pp. 190, with 44 illustrations and 41 tables. Price: Sw.Kr. 25.

Abstracts from Medical Literature.

PATHOLOGY.

Unsuspected Carcinoma of Prostate.

W. C. BAUER, M. H. MCGAVRAN AND M. R. CARLIN (*Cancer*, March-April, 1960) report the discovery of 55 examples of unsuspected carcinoma in 847 consecutive suprapubic prostatectomy specimens. All 55 patients have been followed for a minimum of five years. The crude five-year survival rate was 54%. Twenty-four of the patients were followed for 10 years and the survival rate in this group was 37%. The five-year and ten-year survival rates for patients with well differentiated cancers were 75% and 47% respectively. The same survival rates for those with less than well differentiated cancers were 33% and 14% respectively. The survival rates did not seem to be affected in either group by castration or œstrogen therapy. Therefore the authors suggest that in patients with moderately or poorly differentiated carcinoma discovered after suprapubic prostatectomy a second radical prostatectomy may be a rational approach.

Experimental Iron Overloading.

L. GOLBERG AND J. P. SMITH (*Amer. J. Path.*, February, 1960) report the results of experiments in which they induced a gross degree of iron overload in rats. The siderotic but otherwise apparently normal liver displays an enhanced susceptibility to the action of dietary ethionine, or of a diet poor in protein and vitamin E, or more especially to the two factors acting together. Under this influence, hepatic cirrhosis, which does not supervene in the iron-loaded rat even after two years, makes its appearance within a few weeks, side by side with the massive aggregation of siderophages in the interlobular septa. Lesions in other organs, some associated with lack of vitamin E, are correspondingly accelerated. The most striking of all these instances of synergy is the extensive development of ceroid throughout the body. It is suggested that in the presence of tissue siderosis of such degree there exists a state of "equilibrated swamping" with regard to the iron, the equilibrium being precarious and, in the case of the liver, vulnerable to a host of adverse circumstances. An attempt is made to correlate the various human iron storage syndromes through a common mechanism of origin or development.

Experimental Pigment Cirrhosis.

R. A. MACDONALD (*Amer. J. Path.*, May, 1960), in an experimental attempt to produce hæmochromatosis in rats, has found that a choline-deficient diet which produced cirrhosis of the liver also led to excess absorption of iron and deposition in many tissues. After fatty-liver cirrhosis and iron deposition in various tissues had been induced by feeding choline-deficient diet together with added iron, the animals were fed ordinary laboratory diet for several days or weeks prior to sacrifice. It was found that fat was mobilized from the liver, leaving a portal type of cirrhosis with hæmosiderin deposits analogous to that seen in human pigment cirrhosis. When a normal diet was fed before cirrhosis had developed, iron deposits were found in a non-cirrhotic non-fatty liver, a condition analogous to hæmosiderosis in humans. Autoradiographic studies of DNA synthesis, using tritrated thymidine, indicated that there was no increase in parenchymal cell regeneration in the liver or other organs containing iron. The observations in this study are interpreted as supporting the concept that in humans, hæmochromatosis and hæmosiderosis are variants of one disease.

Siderosis of the Liver.

C. J. UYS *et alii* (*S. Afr. J. Lab. clin. Med.*, March, 1960) have found that on a distributive but not a quantitative basis, siderosis of the liver was indistinguishable in the three racial groups of Cape Town. In the classification used, the more widespread and severe form of siderosis occurred with significantly greater frequency in the Bantu than in the coloured and European people. The coloured and European populations showed an almost equal incidence. The authors regard these findings as favouring the view that dietary habits and iron intake are more important in Bantu siderosis than malnutrition. While association with hepatic fibrosis and siderosis could be demonstrated, there was no correlation with cirrhosis.

Wilson's Disease.

P. J. ANDERSON AND H. POPPER (*Amer. J. Path.*, May, 1960) have reviewed the liver changes in 21 cases of Wilson's disease. Cirrhosis was present in 19 and absent in two. The authors agree with other investigators that fatty metamorphosis precedes the cirrhotic alterations. They also observed glycogen degeneration of nuclei, particularly in the active stage of the disorder, and they incline to the view that this, together with the fatty change, is the underlying abnormality which results in hepatic parenchymal necrosis and stromal collapse, leading to the characteristic post-necrotic cirrhosis.

Primary Aldosteronism.

T. H. CAPERS AND G. J. RACE (*A.M.A. Arch. Path.*, February, 1960) describe a case of primary aldosteronism in which the adrenal glands appeared normal. The patient had rapidly progressing hypertension and sustained hypokalæmia which improved after resection of one entire adrenal and nine-tenths of the other.

Experimental Hypercalcæmia.

F. A. CARONE *et alii* (*Amer. J. Path.*, January, 1960) report that hypercalcæmia of brief duration in dogs, caused by administration of parathyroid extract for only one day, produced structural alterations in the kidneys which were localized by microdissection to the ascending limb of Henle, the distal convolution, and the collecting system. These lesions were of patchy distribution and consisted of epithelial degeneration, calcification and necrosis, often resulting in intratubular obstructing casts. Lesions in the ascending limbs, distal convolutions and cortical collecting tubules usually were healed by regenerating epithelium. Those in the medullary collecting tubules tended to remain as fixed intratubular calcified casts which produced marked dilatation proximally and, in some instances, atrophy of proximal convolutions. Calcification was principally intratubular, not interstitial. Hypercalcæmia of 24 hours' duration produced severe impairment of renal concentrating ability. In most animals there was a drop in glomerular filtration rate and a rise in blood urea nitrogen which was related to the degree of intratubular obstruction. It is postulated that the defect in tubular reabsorption of water is due to the inhibitory effect of excess calcium upon cellular permeability and metabolism. Kidneys with marked structural alterations showed persistent evidence of functional insufficiency. It is stressed that in clinical practice every effort should be made to correct and avoid hypercalcæmia and maintain the serum calcium within the normal range.

Parity and Breast Cancer.

M. T. MACKLIN (*J. Nat. Cancer Inst.*, November, 1959) has found no difference in the incidence of breast cancer among childless women, whether single or married. The highest incidence of breast cancer is in nulliparous women with decreasing incidence paralleling the number of pregnancies. She found also that there is a genetic tendency to breast cancer which is equally as potent as childlessness and that the effects of the two factors are additive. The author discusses underlying factors connected with parity and their relationship to the socio-economic status in the genesis of breast cancer.

Cardiac Conduction Tissue.

G. LUMB AND R. S. SHACKLETT (*Amer. J. Path.*, April, 1960) have made a study of the conduction mechanism of the human heart, with particular reference to the problem of sudden or unexpected death. The interventricular septum was removed from 260 hearts. Serial sections were not found to be necessary, once the structure was recognized. All age groups were investigated. The normal characteristics of the tissue were first established, and then pathological alterations were correlated with abnormalities noted before death. In 35 of these cases lesions were found which were considered to affect the conduction tissues to a significant degree. The authors present an analysis of this group. In 26 of these cases electrocardiograms were available for study, and 25 were examples of sudden death.

Cerebral Vascular Lesions and Peptic Ulcer.

J. B. DALGAARD (*A.M.A. Arch. Path.*, April, 1960) has studied the relationship between cerebral vascular lesions and peptic ulceration. He was not able to demonstrate an ætiological relationship between chronic ulcer and cerebral lesions, but he found that of 208 patients with acute peptic ulceration 32% had a cerebral vascular lesion. His conclusions are that cerebral vascular lesions are the commonest single cause of acute peptic

ulceration as encountered at necropsy. Such acute neurogenic lesions may occur within 12 hours, but are mostly found a few days after the apoplexy. They usually escape clinical diagnosis even in cases in which the complicating ulceration is the immediate or contributing cause of death.

Cardiac Hypertrophy after Coronary Artery Ligation.

T. D. NORMAN AND C. R. COERS (*A.M.A. Arch. Path.*, February, 1960) have investigated the possibility that coronary artery occlusion may cause cardiac hypertrophy. The left coronary arteries of rats were ligated and the hearts of these animals were studied for evidence of hypertrophy six and twelve weeks after the operation by comparison with controls which had been subjected to a sham operation. There was an increase in the weight of ligated hearts at both time intervals. Hypertrophy of the left ventricle occurred only in rats studied twelve weeks after operation, but the right ventricle was increased in weight at both six and twelve weeks after ligation. It is concluded that under certain conditions occlusion of the coronary arteries results in myocardial hypertrophy.

RADIOLOGY.

The Colon in Systemic Sclerosis.

W. T. MESZAROS (*Amer. J. Roentgenol.*, December, 1959) states that involvement of the colon by systemic sclerosis (scleroderma) is probably more frequent than is generally appreciated. Changes would be demonstrated more often if examinations of the large bowel were done routinely in patients with scleroderma. No symptoms may be noted or there may be constipation, diarrhœa, or a combination of these two findings. Abdominal pain may be present. Bleeding from the colon may occur. Occasionally, marked dilatation of the colon is observed, and may be thought to be due to organic obstruction. Involvement of the colon is not necessarily a late finding. The youngest patient found by the author was aged 20 years, with a known history of scleroderma of less than one year. The colon has been reported as atonic, but this is difficult to differentiate from the wide normal variation. Sacculation is due to muscular atrophy and is believed to represent the characteristic change. The saccules contain inspissated fæcal material and are noted most frequently in the transverse colon (inferior surface) and descending colon. Involvement of the sigmoid and ascending colon may also occur. Sacculation is best demonstrated on post-evacuation skiagrams. The normal colon contracts, whereas the atrophic saccules show little or no contraction. These saccules are sometimes referred to as "pseudo-diverticula". They have wider mouths than ordinary diverticula, and can thereby be differentiated. Involvement of the œsophagus occurs in all cases, and changes in the small bowel are found in two-thirds of the patients with involvement of the colon. Demonstration of colon involvement in scleroderma is of practical significance for two reasons: (i) It is important to know that bleeding may occur; this may be due to mucosal ulceration secondary to ischæmia; marked constipation and fæcal impaction may also be a causative factor. (ii) Patients with visceral sclerosis tolerate abdominal surgery poorly. Therefore, if fæcal impaction and atony suggest obstruction, laparotomy is to be avoided until the presence of organic obstruction can be verified. When constipation is present, appropriate dietary and drug regimens are advised to prevent the development of impaction. Barium enema studies in patients known to have scleroderma should be done with care. Over-distention is to be avoided because of the possibility of perforating a thin-walled colon. Similar precautions apply to sigmoidoscopy.

Radiological Appearances of Pulmonary Hypertension.

J. K. MCMYN (*J. Coll. Radiol. Aust.*, June, 1960) has investigated 56 cases of mitral stenosis and noted certain radiographic changes such as the presence of septal lines in the lung fields and the size of the main pulmonary artery and its branches, compared with the pulmonary venous and pulmonary arterial systolic pressures. The conclusions are as follows: (i) The presence of definite septal lines is a valuable sign of pulmonary venous hypertension but not of its degree, except that the pressure tends to be over 20 mm. of mercury. This is consistent with the lines being due to interlobular œdema. (ii) Septal lines also suggest a raised pulmonary arterial systolic pressure, but the converse is not necessarily the case, and in pulmonary arteriolar hypertension alone, pulmonary venous pressure is normal. (iii) Enlargement of the main pulmonary artery indicates pulmonary arteriolar hypertension and gross enlargement suggests a high pulmonary arterial systolic pressure. (iv) There is no correlation between the size of the pulmonary artery and pulmonary venous hypertension.

Small Bowel Pattern in the Zollinger-Ellison Syndrome.

J. M. WEBER, S. LEWIS AND K. H. HEASLEY (*Amer. J. Roentgenol.*, December, 1959) report three cases of the Zollinger-Ellison syndrome—a severe ulcer diathesis associated with non-beta-cell adenomas of the pancreas. In these three cases the small bowel studies showed a distinctive lacy, cobweb-like pattern. At first glance the outline of the small bowel seems smudged and disorganized, but on closer examination a definite pattern is noted. In all cases diarrhœa was a prominent symptom. The authors considered that hypersecretion and hyperacidity alone would not produce the bizarre pattern encountered, since many patients with duodenal ulcer but without pancreatic adenomas secrete large volumes of gastric juice of high acidity yet do not show the pattern observed in the present series. Diarrhœa is not a usual feature of peptic ulceration, but has been observed in at least one-third of the cases of the Zollinger-Ellison syndrome reported to date. It is possible that, in addition to the extreme hypersecretion of acid that is known to occur in the Zollinger-Ellison syndrome, there may also be increased motor irritability with rapid gastric emptying. This might account for the appearance of peptic ulceration distal to the duodenal bulb in upper parts of the small intestine where normally full neutralization has occurred. It would also explain the diarrhœa. The lace-like or cobweb appearance of the small bowel could be accounted for by the same mechanism of hypersecretion and hypermotility. The rapid passage of barium through the bowel, leaving behind a thin film of contrast material to outline the valvulæ conniventes, seems the best way to explain the pattern described. The appearance at times of thickened folds might be either a manifestation of motor disturbance or due to actual œdema of the bowel wall. The variability of the pattern from one X-ray film to another in the same gastro-intestinal study, and the absence of any œdema of the bowel wall at operation or at autopsy favour a motor disturbance rather than œdema as an explanation of the X-ray findings. It is recommended that small bowel X-ray studies be performed in all patients in whom the diagnosis of the Zollinger-Ellison syndrome is being considered. This includes patients with intractable diarrhœa even without peptic ulcer symptoms.

Kinking of the Aortic Arch with Fusiform Aneurysmal Dilatation.

A. F. BARRETT AND G. I. VERNEY (*Clin. Radiol.*, April, 1960) present two cases of kinking of the aortic arch. This is a rare condition and one which causes no disability. It can simulate radiologically lymphadenopathy or tumour of the superior mediastinum, saccular aortic aneurysm or coarctation of the aorta. The kink itself occurs immediately distal to the origin of the left subclavian artery. The aorta ascends to form the arch which is higher than usual, and then descends to the site of the ligamentum arteriosum. There the kink occurs and the aorta then resumes its downward course. In the cases presented the postero-anterior radiographs showed the double contour of the aortic knuckle and the lateral views demonstrated the kink. In order to exclude saccular aneurysm of the arch of the aorta, left lateral tomograms were taken, and these demonstrated that the aortic arch anterior to the kink was of constant calibre.

Retroperitoneal Tumours.

R. A. KEMP HARPER (*Clin. Radiol.*, April, 1960) suggests that in most cases of retroperitoneal tumour there is X-ray evidence of the tumour, if carefully looked for and if the various organs are scrutinized in detail. The roles of plain films and of gastro-intestinal and urinary investigations are discussed. The value of such methods and the information which can be afforded are indicated, and enough evidence can often be obtained to arrive at a diagnosis. The importance of lateral films in these investigations is stressed and there should be resort to more intricate methods of investigation such as aortography and pneumography only when insufficient evidence has been obtained by simpler methods. The features of retroperitoneal tumours and their differential diagnosis are considered and discussed.

Congresses.

THE SECOND ASIAN-PACIFIC CONGRESS OF CARDIOLOGY.

AT its inauguration in Manila, Philippine Islands, in April, 1956, the Asian-Pacific Society of Cardiology accepted the invitation to hold its second Congress in Australia in 1960, the host Society being the Cardiac Society of Australia and New Zealand. Melbourne was selected as the venue for the Congress, and Dr. T. E. Lowe was elected as President of Congress with Dr. Austin Doyle as Honorary Secretary. A strong organizing committee of Victorian members of the Cardiac Society and their wives assured the success of the meeting, which took place between May 29 and June 4, 1960, at the Department of Anatomy, University of Melbourne. Approximately 156 members registered from Australia and New Zealand, and over 70 from other countries. Professor John McMichael, Sims Professor of The Royal Australasian College of Physicians, and Dr. Wallace Brigden of London, R. T. Hall Lecturer of the Cardiac Society of Australia and New Zealand, represented the British Cardiac Society, Dr. F. van Dooren of Brussels the European Society of Cardiology, Dr. G. C. Griffith of Los Angeles the American Heart Association, and Dr. Vittorio Puddu of Rome the International Society of Cardiology, of which he is the Secretary-General. Through the good offices of Dr. Louis Katz of Chicago and Dr. James Watt a group of 12 active American workers in the cardio-vascular field were able to participate, by the aid of a grant from the National Institutes of Health of the U.S. Public Health Service. The Congress was therefore truly international in character, and about 22 nations were represented.

His Excellency the Governor-General of the Commonwealth of Australia, Viscount Dunrossil, graciously offered his patronage to the Congress.

Opening Ceremony.

The opening ceremony, in the Wilson Hall of the University of Melbourne, was impressive and colourful. His Excellency Sir Dallas Brooks, Governor of Victoria, declared the Congress open, and evinced a particular interest in the meeting.

Scientific Programme.

The scientific programme extended over four days, and was conducted in two simultaneous sessions. Ninety-one communications were offered, which had been grouped under regional headings. Papers concerned with the epidemiology of heart disease began with a description by Dr. C. Fortune (Perth) of a ten-year survey of rheumatic fever and rheumatic heart disease in Perth, which has shown a steady decline in incidence, largely due to penicillin prophylaxis. Dr. S. Padmavati (New Delhi) believes that the manifestations of rheumatic fever in her city differ from those in Western countries, as well as producing the majority of cardiac patients found in her clinic. Dr. K. K. Datey (Bombay) confirmed this ætiological spectrum. He found rheumatic heart disease in 22·5%, hypertension in 18·3% and arteriosclerotic heart disease in 14·9%. In a revelatory review of bacterial endocarditis in Sydney during 10 years, Dr. J. B. Hickie referred to the delay in diagnosis, the guises assumed by this complication, and the poor results which depend on slow appreciation of its presence. Dr. K. Somers (Uganda) described the extraordinary endomyocardial fibrosis of Central Africa, for which no single cause has yet been accepted. He inclined to the view that it was a variant of rheumatic heart disease. Dr. W. Brigden (London) gave some clinical pointers to the differentiation of diffuse myocardial disease from constrictive pericarditis, tricuspid valve disease and coronary artery disease. He said that some of these characteristics might be associated with inelasticity of the free walls of the ventricles and enlargement of the septum.

Experimental work related to cardiac surgery was considered in one section. Dr. J. Borrie (New Zealand) gave an account of many studies on hypothermia, vessel grafting and survival of the sheep, and Dr. Currie spoke of the good effects of quinidine in diminishing ventricular fibrillation at low heart temperatures, while the coronary system was being perfused with oxygenated blood. Dr. Simpson and Dr. Gibson (Perth) have used a combination of deep hypothermia and cardio-pulmonary by-pass, with a bubble oxygenator, left atrial cannulation and a heat exchanger. The venæ cavæ are occluded at 15° C., and after surgery, partial by-pass is continued until body temperature returns to normal. Dr. I. Monk (Sydney) has experimented successfully in the reduction of artificially produced mitral incompetence by means of a baffle across the atrium and by alteration in the direction and shape of the pulmonary vein ostia. Dr. Stirling and Dr. Race (Melbourne) have investigated the effects of aortic occlusion and potassium arrest on ventricular function criteria, and concluded that detectable harm follows these operative procedures. Dr. Fanti (Melbourne) gave a warning on the use of silicones to coat containers and tubing; they are potentially irritant to the reticulo-endothelial system, and in any case, unnecessary for platelet preservation. Dr. P. A. Ongley (U.S.A.) reviewed 62 children who developed complete heart block during or after the repair of a ventricular septal defect at the Mayo Clinic. This represented about 17% of all such patients operated upon. Twenty-one died, and in another 15 the heart block was permanent. The complication occurred least frequently when cardiac arrest was not used, and most frequently when pulmonary hypertension was present.

Discussion on the treatment of rheumatic heart disease centred mainly on the surgical relief of the lesion. Dr. W. H. Abelmann (U.S.A.) had studied the clinical and hæmodynamic feature of combined aortic and mitral valve stenosis in comparison with isolated aortic or mitral stenosis. He held that, although symptoms began earlier, prognosis was more favourable than in solitary aortic stenosis. The physical signs of mitral stenosis were less obvious, but were revealed by X-ray examination. The work of the left ventricle was reduced, while left heart catheterization was usually necessary to assess the whole situation accurately. Dr. S. Roy *et alii* (New Delhi) had investigated the left atrial-left ventricular gradient in normal individuals and in sufferers from mitral stenosis by simultaneous readings of pulmonary "wedge" pressures and left ventricular cavity pressures. The normal gradient lay between 2 and 6 mm. of mercury. Dr. D. Stuckey, Dr. Epps and Dr. Monk (Sydney) presented their results relating to 29 patients submitted to aortic valvotomy, and emphasized the value of tomography in delineating degrees of calcification. They stressed the sharp difference between those who had been in congestive failure and those treated earlier. Dr. C. S. Lazatin (Philippines) reported 136 cases in which he had performed mitral valvotomy. He said that in 32 some regurgitation had been recognized, and regurgitation was produced in six patients. Auricular clots were present 24 times, and some valve calcification was found in 19. Only five patients failed to survive the procedure. Dr. H. F. Helmholz, junior, *et alii* (Mayo Clinic, U.S.A.) gave the reasons for errors in diagnosis at catheterization as ignorance of clinical findings, premature decisions taken too early in the procedure, misinterpretation of anomalies, or failure to introduce other tests suggested during the evolution of the laboratory results, and the fact that the usual techniques are sometimes inadequate in complex anatomical deformities of the heart. Dr. E. G. Galea (Brisbane) gave his experience of the rehabilitation of 63 patients followed for some five years. He had decided that the 43 favourable results were related to milder disability, younger age, stable personality and educability, as well as to positive assistance from both employer and doctor.

The section on physiology included numerous interesting papers. Dr. E. Kimura (Tokyo) asserted that generalized coronary insufficiency was associated with *RS-T* depression in the electrocardiogram, while coronary branch insufficiency was associated with *RS-T* elevation, irrespective of the depth of myocardium involved. Dr. G. M. Maxwell (Adelaide) explained that dopamine, a precursor of noradrenaline, caused increased cardiac output and powerful coronary vasodilatation. Dr. S. Hayase (Kyoto), working with anæsthetized dogs, found that sympathetic nerve stimulation augmented coronary flow and the uptake of oxygen. Small amounts of sympathomimetic drugs were more effective than large doses. He thought that the "coronary artery insufficiency" pattern of the electrocardiogram was an expression of increased myocardial efficiency. Dr. M. Toor (Israel) had further investigated the effects of serotonin in the pressure responses of various cardiac chambers and pulmonary vessels. The pulmonary veins showed the greatest response. Dr. H. V. Hodge and Dr. E. G. McQueen (New Zealand) seem to have shown that in rats, when unilateral renal ischæmia was produced and hypertension was prevented by reserpine, cardio-vascular damage was less in general, but increased sodium intake was high. Dr. R. F. Rushmer and others (Seattle, U.S.A.), in a striking contribution,

described the similar regional adaptations in blood flow and left ventricular performance on exercise and on stimulation of certain areas of the diencephalon. Ultrasonic flowmeters placed in peripheral and visceral arteries showed that the renal blood flow remained stable under these conditions.

The section on coronary and arterial disease was opened by Sir Charles Burns (New Zealand), who gave the clinical characteristics of 500 patients with ischæmic heart disease seen in private practice. Dr. H. M. Whyte and Dr. R. B. Goldrick held that the proneness of the obese to ischæmic heart disease could be related to increased serum beta lipoprotein content, increased body bulk leading to raised blood pressure and delay in fibrinolysis. Dr. E. H. Roche (New Zealand), in a review of the electrocardiograms of 12 patients with subendocardial infarction who came to autopsy, found *S-T* segment depression and inverted *T* waves in left ventricular leads in all, but abnormal *Q* waves only twice. Dr. J. Stokes III and co-workers (Boston, U.S.A.) described higher incidence of coronary artery disease and high serum cholesterol levels in the spouses of subjects with angina pectoris and myocardial infarction than in controls in the Framingham study; this suggested an environmental factor, as opposed to the incidence of hypertension, in which genetic effects may be more influential. Dr. K. Ishikawa (Tokyo) has been able to distinguish occlusive changes from vasospasm in finger-tips and toe-tips by means of photoelectric plethysmography and temperature records, before and after body warming. Dr. A. J. Barnett and Dr. K. Morris (Melbourne) critically reviewed 62 cases of arterial grafting by various techniques, concluding that less than 10% of patients were suitable for operation, and that only 50% of grafts continued to function after three years. Dr. H. B. Windsor (Sydney) defined the factors influencing surgical management of intrathoracic aortic aneurysm, using woven "Teflon" for reconstruction, partial by-pass for aneurysms beyond the subclavian artery, and total by-pass for those proximal to it.

Problems of the pulmonary circulation were considered by Dr. A. Jose (Sydney), who had estimated pulmonary blood flow and volume as the difference between the dispersion of indicator dye injected into the left atrium and pulmonary artery, followed by sampling at a common point. The normal pulmonary blood volume varied between 126 and 598 ml. per square metre of body surface, and was related to ventricular stroke volume. There was an inverse relationship between pulmonary blood volume and pulmonary vascular resistance, suggesting that both pulmonary veins as well as pulmonary arteries were less distensible in patients with high vascular resistance in the lungs. Dr. G. Sloman and Dr. B. Gandevia (Melbourne) have successfully applied estimates of ventilatory capacity, exercise tolerance and ventilatory response to exercise to 20 patients before and after cardiac surgery, and related these to clinical, radiological and hæmodynamic findings. They believe that a simple test of ventilatory response to exercise can be used as a routine measure for objective assessment. Dr. H. J. H. Colebatch and Dr. D. Halmagyi (Sydney) showed, by controlled respiratory experiments in sheep, that mean pulmonary artery pressure and resistance rose during expiration, presumably owing to an increase in alveolar pressure, and discussed the significance of this mechanism in emphysema. Dr. W. A. Seldon (Sydney) concluded that a mean pulmonary capillary pressure of greater than 20 mm. of mercury together with a *v* wave greater than 15 mm. contraindicated mitral valve surgery in mixed mitral valve disease.

Dr. W. R. Milnor (Baltimore, U.S.A.) took up the question of vascular distensibility in the lesser circulation and related it to vascular volume, pressures and flow in dogs and man. He held that the distensibility component could be a controlling factor, additional to myocardial efficiency in the determination of stroke output. Dr. B. Tofler (Perth) recommended auscultation of the right side of the chest in deep inspiration in order to discriminate between mitral and tricuspid systolic murmurs. Dr. T. V. O'Donnell and Dr. M. McIlroy (San Francisco), after investigating the hæmodynamic benefit of squatting in cases of tetralogy of Fallot pathology, concluded that increased central blood volume, improved pulmonary blood flow and better left ventricular filling combined with decreased right-left shunt, following increased systemic resistance, to produce improvement. Dr. M. Toor and colleagues (Israel) had studied the effects upon the electrocardiogram of severe exercise in a hot climate under varying conditions of hydration and noted slight changes related to electrolyte disturbance occurring in such environment. Dr. J. M. Gardiner (Melbourne) reviewed the phonocardiographic features of innocent systolic murmurs. He stated that it was probable that several types were due to a minor anatomical abnormality. Dr. E. Braunwald (Bethesda, U.S.A.) described satisfactory results in left heart catheterization by the transseptal route. A catheter hiding a sharp metal needle is passed into the fossa ovalis from the femoral vein and readily perforates the septum at this point, permitting introduction of the catheter tip into the left atrium. Dr. M. G. Taylor (Sydney) discussed the changes in the form of the central aortic pulse due to reflected waves from the periphery, which are of the magnitude of 30% to 40%. Further work on reflections from head, neck and viscera is planned. Dr. P. J. Korner (Sydney) has quantitated pulmonary incompetence in dogs by means of a flowmeter and dye dilution, and was satisfied that variations due to varying circulatory dimensions could be corrected by examination of the abnormal curves themselves. Dr. H. J. Ueda (Tokyo) has determined cardiac output by external scanning, using serum abumin labelled with ^{131}I as the indicator injection. His results correlated well with those obtained by the direct arterial sampling method. Aminophylline appeared to decrease cardiac output in the presence of congestive failure. Dr. N. Kimura (Japan) concluded that the vector cardiogram was superior to the conventional electrocardiogram in the detection of left ventricular hypertrophy.

Lipids and thrombosis were naturally the interest of several speakers. Dr. J. D. Hunter (New Zealand) had investigated the fasting blood cholesterol levels in 1000 presumably normal males, and observed a gradual rise from 190 mg. per 100 ml. at the age of 20 years, up to 240 mg. per 100 ml. after 40 years. The relationship of such levels to various environmental and physical factors, and in comparison with sufferers from coronary artery disease, was presented. Dr. R. B. Goldrick (Sydney) said that in New Guinea natives, fat provided 18% of the daily calories, and that these natives had significantly lower blood levels of cholesterol and lipoprotein. The dispersion of cholesterol among the lipoproteins was more uniform in the natives than in white Australians of comparable age, in whom most cholesterol was found in the beta lipoprotein. Native blood clotted more rapidly, but fibrinolysis was also very rapid. Dr. M. Murakami (Japan) had found low concentrations of heparin-like substances in sufferers from arteriosclerosis and its complications. There was an inverse relationship between the endogenous clearing activity and the serum beta lipoprotein level. Dr. P. J. Nestel and Dr. C. Hollett (Melbourne) had come to similar conclusions. They pointed out that the reduction in clearing activity appeared to be due to unrelated platelet concentration, but a change in optical density preceded clearing, and that was more obvious in platelet-poor serum. Platelets inhibited physical clearing significantly more than chemical clearing. Dr. J. Shimamoto (Japan) showed clearly by a cinematographic film that endothelial damage with removal of platelets in animals followed the administration of a variety of macromolecular substances. Repeated administration led to permanent arterial disease, with stainable subendothelial fat. Dr. G. V. Hall and colleagues (Sydney) reported the results of their observations on the handling of labelled fat by patients with coronary disease. They held that clearing of this fat was significantly slower in 80% of such patients. Anticoagulants, both natural and artificial, improved the situation. "Stypven" clotting times were also investigated in both patients and controls. Dr. R. J. Havel (San Francisco) has noted that, although triglycerides are removed more slowly from the blood-stream in subjects with coronary artery disease, the resultant products of this enzymatic action—namely, glycerol and fatty acids—do not increase in concentration. A true enzymatic deficiency appears to exist in idiopathic hyperlipæmic states, but in other diseases in which hyperlipæmia exists, it is more probably due to over-production of triglycerides in the plasma than to defective removal. Dr. R. S. Gordon (Bethesda, U.S.A.) described a protein-losing gastroenteropathy associated with cardiac failure. Sir Horace Smirk (New Zealand), in opening the section on high blood pressure, concluded from cross-transfusion experiments from hypertensive rats that the raised blood pressure in those animals was maintained by a neural rather than a humoral mechanism. Dr. K. V. Gour (India) has investigated the relative incidence of hypertension in rural and industrial workers in Kanpur, India, and found a higher incidence in the latter. Dr. K. H. Cho *et alii* (South Korea) has established normal casual blood-pressure figures from 30,000 South Koreans. Dr. R. F. Whelan *et alii* (Australia) demonstrated a direct vasodilator effect in the skin of animals receiving intraarterial injections of reserpine. The response was not mediated through nervous

channels, and could be reversed by noradrenaline. Dr. G. E. Bauer (Sydney) said that bretylium tosylate ("Darenthin") was free from parasympathetic side effects, and was a satisfactory hypotensive agent. Dr. McCubbin (Cleveland, U.S.A.) described experiments in which he had estimated electrical activity in pre-ganglionic and post-ganglionic sympathetic nerve fibres after administration of various hypotensive agents. He said that tetraethyl ammonium and mecamylamine acted on peripheral ganglia, while reserpine acted centrally like serotonin and noradrenaline, so that a fractional dose given into a cerebral ventricle had as much effect as a full dose given intravenously. Those effects were opposed by central administration of vasodilator drugs, and might depend on local changes in blood supply. Guanethedine hypotension resulted in part from the ability of that drug to prevent peripheral release of noradrenaline. Dr. McCubbin thought that the sensitivity of the carotid sinus mechanism could be "reset" by prolonged changes in blood pressure.

Further contributions concerned with myocardial metabolism included a communication by Dr. T. Kobayashi (Japan), who is engaged in a study of coronary sinus blood in sufferers from hypertension, coronary sclerosis and diabetes mellitus. Dr. W. G. Naylor (Melbourne) believes that a cell-membrane permeability factor is the common mechanism for the inotropic action of cardiac glycosides, certain steroids, electrolytes, etc., and that this factor is probably under hormonal regulation. By the use of paper chromatography and isotopic labels, Dr. S. E. Wright (Sydney) has examined the behaviour of the available cardiac glycosides in regard to their absorption and disposal. He believes that these can be related to the polarity of the molecule as well as to the chemical structure. Dr. G. C. Griffith (Los Angeles, U.S.A.) reported studies in the concentration of trace metals in the myocardium in health and disease. In myocardial infarction, tissue manganese concentration falls sharply, while the plasma copper level rises. In advanced coronary artery disease, calcium values are low. Hypertensive patients have increased lead and cadmium in their cardiac tissues. Professor W. V. Macfarlane (Canberra), after investigation of the action potential and refractory period of various sites in the amphibian heart, postulated that these varied according to oxygen supply and high energy phosphate resources, this dependency being greatest in the conus and least in the sinus area. Dr. M. J. Rand (Sydney) referred to the presence of cholinergic fibres in the heart, and suggested that the acetylcholine released after their stimulation in turn released noradrenaline from its stores, and that was in effect adrenergic.

Communications concerned with medical aspects of congenital heart disease included some important papers. Dr. K. Soekarjo (Indonesia) produced a case report of a boy with a mediastinal teratoma. Dr. J. B. Lowe (New Zealand) spoke of the diagnostic and surgical difficulties met with in atrioventricular canal defects, particularly in regard to the occasional absence of a systolic apical murmur and a left-sided electrocardiogram, with laboratory evidence of an intraventricular shunt. He said that rare association with pulmonary stenosis, tricuspid incompetence or cardiomyopathy might make the situation very complex. Dr. K. I. Vytilingam (India) spoke of the spectrum of aortic root deformity with ventricular septal defect, which has been called the tetralogy of Fallot, and still advocates the anastomotic operation in severe cases. Dr. J. F. Farrar and colleagues (Sydney) described 15 infants with primary pulmonary hypertension, with 11 autopsies. Death occurred either early or after two years. The bronchographic and histological appearances were illustrated excellently. A trial with antiserotonins was planned. Sir Alexander Murphy (Brisbane) described the clinical and radiological features and differential diagnosis of three patients with agenesia of the left main pulmonary artery. Dr. I. S. Wallman (Perth) had found a large patent ductus arteriosus in eight babies whose mothers had contracted rubella early in pregnancy during the 1958 epidemic. Five babies required operation under the age of five months. Dr. S. B. Roy (India) referred to a study with Dr. J. Dickson and others in Boston, U.S.A., directed to evaluation of prolonged veno-arterial pumping to relieve cardiac embarrassment following reversible accidents such as myocardial infarction or pulmonary embolism. Blood is withdrawn by gravity from the superior vena cava and pumped into the aorta. Satisfactory perfusion of all vital areas has been maintained clinically for up to 26 hours.

Discussions concerning the surgery of congenital heart disease revealed some interesting figures. Dr. B. G. Barrett-Boyes (New Zealand) described excellent results in simple atrial septal defect using the "atrial well" technique of Gross. Dr. R. Nicks (Sydney) defined the various types of primum atrial defects as encountered in pathological specimens and in 14 clinical cases. He agreed with Barrett-Boyes that their surgery required the extra-corporeal circulation. Dr. K. Morris (Melbourne) referred to the difficulty in choosing, for surgical relief, the patient with simple aortic stenosis unaccompanied by aortic incompetence or coronary artery disease. He had performed 40 operations by direct vision, using a combination of hypothermia with halothane anæsthesia and partial cardiac by-pass. Dr. L. Sauvage (Seattle, U.S.A.) has found that pericardial autografts function well for vessel replacement, and for widening the outflow tract of the right ventricle. He is satisfied that with the aid of left heart by-pass, this membrane can be used for reconstruction of the mitral valve. Dr. P. Ongley (Rochester, U.S.A.) described the diagnosis of corrected transposition of the great vessels, after an experience of 33 cases. The anomaly is associated with displacement of the left atrio-ventricular valve, partial or complete heart block, and ventricular septal defect with low *R* waves in Lead V_1 and presence of *Q* waves in Lead V_1 but not in V_6. On X-ray examination the pulmonary trunk is not obvious, but the left atrium is large. Cardiac catheterization permits localization of the displaced atrio-ventricular valve, and difficulty is met with in entering the pulmonary artery. Selective angiography from the right ventricle is diagnostic. The importance of differentiating this anomaly from simple ventricular septal defect is obvious. Dr. A. W. Venables (Melbourne) had seen in a period of 18 months 29 infants aged under 12 months with patent ductus arteriosus, alone or complicated. He emphasized the necessity for thoracotomy in a baby in heart failure with a wide pulse pressure, pulmonary plethora, left ventricular over-activity and a murmur compatible with patent ductus. Dr. C. J. O. Brown and colleagues (Melbourne) reviewed 72 patients with coarctation of the aorta who had had the coarctation resected. Six required homografts and two a subclavian by-pass. There were six deaths in the series. In a further group of papers on coronary vascular disease, Dr. E. P. George (Sydney) interpreted the lipid tolerance curve obtained by the isotopic trace method described above by Dr. G. V. Hall *et alii*. Dr. A. G. Sison (Philippines) gave the results of a survey of 10,702 autopsies, in which an association between gall-bladder disease and arteriosclerosis had been sought. Only 9·6% of males and 15% of females with arteriosclerosis had gall-stones. While the over-all incidence of arteriosclerosis was high (41·9%), the incidence of coronary artery disease was low. Dr. C. McRae (Melbourne) referred to the diagnosis of ventricular paroxysmal tachycardia from supraventricular tachycardia with aberrant intraventricular conduction, and concluded that ventricular capture by supraventricular impulses and ventricular fusion beats were the most reliable electrocardiographic signs of this arrhythmia. Dr. J. G. Sloman (Melbourne) described his method of coronary angiography by injection of dye into the base of the aorta through a large catheter with side holes, the actual injection being triggered by the *R* wave of the electrocardiogram so as to fill the coronary vessels during diastole. The method also allows some appreciation of the state and function of the aortic valve in patients with aortic stenosis. Dr. J. M. Agar (Geelong) reviewed 1000 patients whom he had treated for coronary artery disease. Half the group suffered from myocardial infarction, and half of this sub-group have died, as compared to one-quarter of the ischæmic sub-group. Gall-bladder disease, peripheral arterial disease and obesity did not appear to influence the mortality rate. Dr. W. Laurie and Dr. J. W. Woods (Australia) believe that the course and outcome of coronary artery disease depend more upon the efficiency of congenitally acquired coronary artery anastomosis than upon factors operating later in life. Dr. R. Heilig (India) advocated the exposure of individuals predisposed to coronary artery disease to gradually decreasing oxygen pressures in a low-pressure chamber as a stimulus to the development of coronary artery collateral circulation.

In the final section on heart failure, Dr. T. E. Lowe (Melbourne) emphasized the role in the genesis of cardiac œdema of physical factors controlling the partition of fluid in the three body compartments. This follows mechanical laws in response to the activity of a diencephalic centre for volume control. Dr. M. Mackawa (Japan), from studies of renal sodium clearance in cardiac œdema, found an inverse correlation between this parameter and peripheral venous pressure, supporting the "forward" theory of cardiac failure. Dr. R. Gunton (Canada) found from hæmodynamic observations on patients in shock after myocardial infarction that cardiac output was reduced by half and central blood volume was increased. Other patients with hypotension, but not in shock, were in

peripheral vascular failure. Vasopressor drugs used in the treatment of 50 shocked patients failed to reduce the 80% mortality. Such patients might have benefited by temporary mechanical assistance to the circulation. Dr. E. V. Newman (Nashville, U.S.A.) said that it was impossible to separate hydraulic from metabolic events in congestive heart failure. Vigorous diuretic therapy might ameliorate primary hydraulic manifestations although directed to a metabolic fault such as sodium retention. If those failed, reduction of serum aldosterone level by spirolactones, which specifically blocked the action of aldosterone on renal sodium retention and potassium loss, might rescue some patients from intractable cardiac failure. Dr. R. S. Ross *et alii* (Baltimore, U.S.A.) measured capillary permeability in the œdematous limbs of cardiac patients by retrieving labelled serum albumin, injected intravenously from Southey's tubes. In normal subjects, higher plasma volumes increased the loss of the indicator substance, but not in the presence of congestive heart failure. The transcapillary exchange rate was less than the mean rate of exchange throughout the body. Professor J. McMichael (London) illustrated the striking effect of digitalis upon the right and left ventricle in failure. He said that this inotropic action bore no relationship to ventricular hypertrophy, and might disappear in advanced failure. Control of heart rate played a minor role. Digitalis toxicity was enhanced by potassium loss. That could occur in advanced failure or after the use of powerful diuretics. Digitalis had a directly beneficial effect on the thyrotoxic heart, apart from the lowering of ventricular rate in auricular fibrillation.

Conclusion of Congress.

The closing remarks were made by Dr. J. K. Maddox as President of the Asian-Pacific Society of Cardiology. He expressed the feelings of all participants that they had enjoyed a week of profit and pleasure. The Congress, the first International Medical Congress to be held in Australia, had been a stimulating experience, and had been excellently organized by the Melbourne committee. On behalf of the Society, he thanked all those men and women of Melbourne who had worked so successfully, the University authorities, the technicians, the students, and the drug firms who had helped in many ways. He expressed gratitude to the speakers and chairmen, and to the panellists and exhibitors. He made particular reference to the assistance given by the Governments of Australia and New Zealand, the R. T. Hall Trust, Dr. Louis Katz and the International Society of Cardiology Foundation, the Public Health Service of the U.S.A., and the Trustees of the National Art Gallery and many others. He stated that the work of the Society would continue between congresses, by means of its Research and Education Committees. The Society had accepted the invitation of the Japanese Circulation Society to hold its next congress in Japan in 1964. In conclusion, Dr. Maddox said that he thought that the good results of the Congress did not end on the final day, but continued indefinitely in new friendships and understanding between cardiologists and others within the Asian-Pacific area. He then invited Dr. T. E. Lowe, President of the Congress, formally to declare the Congress closed. Dr. Lowe, in doing so, expressed the gratification of the Organizing Committee on the success of the meeting, and on their behalf wished all a safe return and an early reunion.

General Assembly of Asian-Pacific Society of Cardiology.

The General Assembly of the Asian-Pacific Society of Cardiology met on Monday, May 30. The Society now comprises the following members: Australia and New Zealand, India, Indonesia, Iran, Israel, Japan, Pakistan, Philippines, Republic of China (Taiwan), and South Korea. An application has been made by Hawaii. Individual members have been accepted from Hong Kong, Malaya, New Caledonia and Thailand.

The office-bearers of the Society for the period 1960 to 1964 are as follows: *President*, Dr. J. Kempson Maddox (Australia); *Immediate Past President*, Dr. Antonio Samia (Philippines); *Vice-Presidents*, Dr. Magojiro Maekawa (Japan), Dr. R. Khambatta (Pakistan), Dr. J. B. Nolasco (Philippines), Dr. S. Padmavati (India); *Secretary and Treasurer*, Dr. James M. Gardiner (Australia).

A number of committees were set up, including a Research Committee under the chairmanship of Dr. Malcolm Whyte of Sydney. A research programme is being mapped out, in cooperation with the World Health Organization.

An invitation was extended by the Japanense Circulation Society to hold the third Asian-Pacific Congress of Cardiology in Japan in 1964, and this has been accepted.

Out of the Past.

THE MELBOURNE HOSPITAL, 1854: FINANCES.

[From the Seventh Annual Report of the Committee of Management of the Melbourne Hospital, 1854.]

IT will, doubtless, be within the recollection of many of the Governors and Contributors to the Melbourne Hospital, that in concluding their Report for the year 1853, the Committee of Management (after deploring the state of the finances, the outlay being in excess of the income, and the prospect of increased expenditure, with a diminishing revenue) stated their confident conviction that all assistance, pecuniary or otherwise, which might be required would be abundantly supplied.

With feelings of profound gratitude to Almighty God, the gentlemen, to whose care has been confided the management of the Institution during the past year, testifying that the confidence thus expressed has been more than realised, for not only has the debt referred to been liquidated, but the maintenance account, although increased upwards of 33 per cent, has been paid, and a small balance carried forward into the present year. In addition, your Committee announce the completion of the centre building (except in some minor particulars), at the cost of upwards of £13,000, and its fitness for occupancy, so soon as funds are available for the purchase of outfit etc. The erection of this building will enable your Committee to accommodate 124 additional patients, which number may, under extraordinary circumstances of pressure, be increased to 150; besides affording a spacious Committee Room, apartments for the use of the Resident Surgeon, a Dispensary, receiving-rooms for patients, operating and two bath rooms.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

A MEETING of the Pædiatric Society of Victoria was held at the Royal Women's Hospital, Melbourne, on October 14, 1959.

Cerebro-Spinal Fluid Findings in Normal New-Born Infants.

DR. F. FORSTER said that some five years previously he had undertaken an investigation of the cerebro-spinal fluid findings in normal new-born infants. The reason for doing so had been that as a resident, assistant pathologist, and pædiatric and obstetrical registrar at the Royal Women's Hospital, he had come to believe less and less in the then-quoted norms for the cerebro-spinal fluid in the new-born. For example, a protein content in excess of 100 mg. per 100 ml. had been regarded as being indicative of a pathological state, and a figure of 160 or so had been looked upon with concern. When such a level was reported, it had been not uncommon for the neonatal pædiatrician to diagnose cerebral hypoxia and to order repetition of the lumbar puncture. Often by that time the baby had clinically recovered from his earlier troubles; but that had not stopped repetition of the procedure, sometimes several times, for there had been a feeling that such a protein level indicated something wrong, and that something ought to be done about it. Ultimately a thriving baby with a protein level slowly falling, but commonly still above 100 mg. per 100 ml., was discharged from hospital but brought back to the follow-up clinic. A further stimulus to the investigation had come from a sister-hospital, where a neonate, whose cerebro-spinal fluid white cell count was a little over 40 per cubic millimetre, had provoked considerable interest. That baby had had a very considerable work-up, and when all studies gave negative results, the final conclusion, with no clinical support, had been that the trouble was probably viral. Finally, when he (Dr. Forster) had turned to the literature for help on the normal cerebro-spinal fluid, he had found a morass of contradictory statements.

Dr. Forster went on to say that in his investigation the lumbar puncture had always been performed with the baby on his side and with a standard needle, and usually only one attempt was made at placing the needle in the right spot. The slightest evidence of traumatic blood-staining meant rejection of the specimen. Microscopic examination

of the cerebro-spinal fluid of the new-born almost invariably showed a number, often considerable, of red blood corpuscles, and just what was an acceptable upper limit was debatable. For no good reason, he had set that at 500 per cubic millimetre, and no specimen showing more had been included in the report. With the needle correctly inserted, there had usually been a good flow of fluid, and only two to three millilitres had been removed. No specimen had been obtained by aspiration. His success rate at obtaining a sample which was satisfactory by those criteria was about 50%. By a normal new-born baby he meant one who was born at full term, whose mother had had no significant complication of pregnancy, a normal labour and a spontaneous delivery, and whose progress was normal whilst in hospital. The only thing which set those babies apart from others was that their mothers were in most cases not married. There had been no complications arising from the investigation.

The report was based on the results obtained from 40 babies. Their ages ranged from three hours to five days. The cell counts were made by one person, and the protein determinations also by a single individual. The protein content had been estimated in duplicate by the micro-Kjeldahl technique, and at the same time a flocculation test had been performed. The latter, with a few exceptions, had given a fair approximation to the result obtained by the micro-Kjeldahl method.

Dr. Forster said that he had found that xanthochromia was present in 40% of specimens. That determination had been purely subjective, and sometimes there had been disagreement between himself and other observers. The protein content had varied from 50 to 300 mg. per 100 ml. The results formed a symmetrical distribution curve with the mean and mode falling at about 160 mg. per 100 ml. The mean of 11 determinations performed in the first day of life was 190, and for nine determinations on the fifth day, 150. The white-cell count had ranged from 0 to 40 per cubic millimetre, with a mean of 13·5. The distribution of cell types had been: lymphocytes, range 0 to 25, mean 7·7 per cubic millimetre; monocytes, range 0 to 21, mean 4·2 per cubic millimetre; polymorphs, range 0 to 9, mean 1·6 per cubic millimetre. Those results indicated the great variations in the cerebro-spinal fluid of the new-born, variations which were only to be expected when one considered the differences seen in labour and delivery.

DR. J. COLEBATCH said that whilst those babies were apparently normal, it was still possible that some were not. There was an analogy with observations of the hæmoglobin level of apparently healthy school-children. It was possible that some of the babies studied had suffered from anoxia or subclinical virus infection. Dr. Ashley Weech had quoted 15 cells per cubic millimetre as the upper limit of normal.

DR. KATE CAMPBELL said that it was sometimes said that new-borns had very little cerebro-spinal fluid, but Dr. Forster had shown that that was not so. She thought that the different values quoted from different countries might reflect differing obstetric standards.

DR. R. SOUTHBY asked whether all the mothers were primiparæ.

Dr. Forster replied that that was not so.

DR. B. NEAL asked whether there was any correlation between xanthochromia and protein levels.

Dr. Forster replied that he had found no correlation. In concluding the discussion, he said he thought that the investigation had been worthwhile, in that it had led to the omission of many unnecessary lumbar punctures.

Amniocentesis in Rh-Immunized Mothers.

DR. E. MACKAY said that isoimmunization of the pregnant woman was responsible for a small but increasingly significant proportion of perinatal deaths. The condition was of particular importance to the individual patient, as it might occur in each pregnancy, often in an increasingly severe form. Apart from immediate mortality, there was considerable evidence that permanent disability might ensue in surviving infants. In recent years, advances in the treatment of erythroblastosis fœtalis had been mainly concerned with management of the infant after delivery. Refinements in the technique of exchange transfusion and the employment of multiple transfusions when necessary had saved many babies from death or disability. The place of premature induction of labour had not, as yet, been finally settled. Several well-known workers in that field had abandoned the procedure, believing that the hazards of prematurity more than offset the increase in the number of living infants produced. Recently, with improvements in the management of the premature infant and greater experience in multiple exchange transfusions, there had been a trend back to premature induction of labour. The emphasis was now on termination of pregnancy only in selected cases, in which the prognosis was known to be poor if the infant was left in utero. It was in the selection of such cases for early induction of labour that amniocentesis might be of value.

Dr. Mackay went on to say that in general the prediction of the Rhesus status of the infant in Rhesus immunization was made on a study of the history of the patient, serological testing and amniocentesis. In a varying degree, those would also indicate the intensity of the hæmolytic process. Unfortunately none of those methods was by any means absolute. The history was most helpful if there had been a Rhesus-negative infant produced. That would establish that the father was heterozygous for the Rhesus factor. Testing of the father's blood alone would only indicate probabilities in that respect, being based on population statistics. The history might also indicate the degree of severity, as many patients tended to be similarly affected in succeeding pregnancies. There were, however, a number of cases in which a mildly affected infant would be followed by an infant with hydrops, and rarely the reverse might happen. In the serological study of Rhesus isoimmunization, the main advance had centred on the use of the indirect Coombs technique for the detection of maternal antibodies. That had been found to be more reliable than the older saline and albumin techniques, and gave a reasonable correlation with the severity of the disease. Unfortunately there were exceptions to that rule; severely affected babies were sometimes born when the titres had not been ominously high, or when there had been no rise or even a fall in levels.

It was for that reason that amniocentesis had been employed in isoimmunized pregnant patients. Two workers in England had reported on their experiences in that field—namely, Bevis and later Walker. In 1956, Bevis had reported the investigation of 71 specimens of liquor from 40 sensitized Rhesus-negative women. He had employed multiple punctures from the twentieth week, in some cases up to six taps being obtained. Including a previous series, a total of 150 patients had been examined without incident, the only exception being an abortion of an abnormal fœtus at 24 weeks, two days after paracentesis. That worker had noted that the spectral absorption curve offered a reliable guide to the ensuing clinical state—in particular to whether the baby was hydropic or would manifest mainly jaundice or anæmia. Levels for bilirubin and oxyhæmoglobin in the liquor had been established, and it had been found that high oxyhæmoglobin and bilirubin levels tended to be followed by kernicterus, whereas a rise in bilirubin level alone was associated with a hæmolytic anæmia. Bevis had noted that the hæmolytic process might occur quite suddenly, presumably corresponding to Kelsall's "episodes of immunization"; but in general an early appearance indicated a worse prognosis. It had been noted that severe hæmolyses before the thirty-second week invariably presaged a fatal outcome.

If the cord serum was examined, the findings were essentially the same as in the liquor amnii, except that hæmoglobin and bilirubin concentrations were far greater—for instance, 7·1 and 1·08 as opposed to 0·95 and 0·16 mg. per 100 ml.

Walker had described his experience in 74 cases. The predictions had been correct in 17 of 19 Rh-negative babies, and in 63·6% of cases in which the baby was affected. In the investigation of the false-negatives, it had been noted that if tests made prior to the thirty-fifth week only were studied, the correct prediction rose from 63·6% to 92·5%, indicating that conflicting curves could be obtained before and after that time. That worker had postulated that the more mature baby might be better able to deal with the excess bilirubin.

In the Royal Women's Hospital, amniocentesis was performed on most isoimmunized patients at about 32 to 33 weeks' gestation. A further specimen of amniotic fluid was obtained at delivery. Both specimens were examined spectrophotometrically. Macroscopically, the fluid obtained varied from the normal clear or milky liquor to one deeply yellow in colour. Dr. Mackay showed specimens from three consecutive taps on different patients, demonstrating the range of colour and spectrophotometric curves found. In each case, the findings were suggested by the history of the previous pregnancies. The patient with the most deeply-stained liquor had borne one normal infant, and had then experienced three consecutive fœtal deaths due to erythroblastosis. Already the present fœtus had succumbed on the day chosen for induction of labour at 33·5 weeks. The moderately-stained liquor was from a patient who had nine previous pregnancies, the last four requiring exchange transfusions; the most recent of the four had been given three exchanges. The least-pigmented liquor was from a

patient who had had immunized pregnancies, but whose previous infants had required no treatment.

The antibody titres of these patients, assessed by both the indirect Coombs and papain treated cell methods, were then shown and compared with the information given by the pigment studies. A further case was then shown of a patient who had lost her first child from preeclampsia. The next two children were normal and the latest had required an exchange transfusion. At eight weeks in the present pregnancy the antibody titres were, albumin 512, saline 0, indirect Coombs 256. Serological testing of the husband and children confirmed that he was heterozygous. At 30 weeks the indirect Coombs titre had risen to 512 and at 32 weeks there was a further rise to 1024. Amniocentesis at this time produced a clear liquor and the patient was allowed to come into spontaneous labour.

Another sphere of usefulness of the procedure lay in the assessment of patients who had been referred in the later months of pregnancy with inadequate antibody tests having been performed (i.e., the indirect Coombs or possibly the papain method). In those cases, there was no opportunity to study the behaviour of the titres over the whole pregnancy and it was probable that the information gained from the liquor would be of more help in such cases.

In more recent months, studies on the protein content of the liquor amnii had been undertaken. In the three patients discussed previously, it had been noted that there was a progressive increase in the amount of protein present on the electrophoretic strips with increasing severity of the disease. The protein was composed mainly of albumin, and the pattern was somewhat similar to that seen in the urine in the nephrotic syndrome; increase in permeability might be a factor in the appearance of protein in the liquor.

Apart from a prediction of the best time for induction, there was some indication that help might be given in deciding the need for exchange transfusion. This could best be done by a study of the values of oxyhæmoglobin and bilirubin in the serum as suggested by Bevis and Abelson, but liquor interpretation would give some indication.

DR. KATE CAMPBELL said that amniocentesis was more helpful than antibody titre levels, which might be upset by anything that affected the mother's immunological response.

DR. M. POWELL asked for details of the technique of amniocentesis.

Dr. Mackay replied that the tap was usually performed by inserting under local anæsthesia a 22-gauge needle into the midline at the thirty-second week of pregnancy. At that time the fundus was above the umbilicus and with experience one could nearly always obtain fluid.

MR. A. M. CLARKE asked whether the procedure was of value in any other conditions.

Dr. Mackay replied that it was of some use in diagnosing renal agenesis and placental insufficiency.

DR. VERA KRIEGER said that the main difficulty in interpreting titres of antibodies occurred in those cases in which there was a high titre from a previous pregnancy. There had to be at least two tubes difference in titre before significance could be attached to a rise or a fall.

DR. J. SHELTON said that he did not think that amniocentesis would take the place of serological investigation. Walker would never interfere in any case where the father was heterozygous. It had to be remembered that there was a failure rate in amniocentesis, although this was admittedly low in expert hands. The erthroblastosis clinic at the Royal Women's Hospital was one of the largest in the world, handling about 150 cases a year.

Antibiotic Resistant Organisms.

DR. HILDRED BUTLER gave a paper on antibiotic resistant bacteria in a maternity hospital, which was the only sort of hospital which admitted bacteria-free patients. These were usually colonized from their mothers, but also from the hospital environment which would always contain pathogenic bacteria, some of which were antibiotic resistant. Even when disease was not produced this had the effect of babies leaving the hospital as carriers. In an endeavour to meet this problem an infection-control sub-committee had been set up at the Royal Women's Hospital. In 1957 the majority of staphylococci cultured had been tetracycline resistant. It had been recommended by the sub-committee that at any one time only one antibiotic was to be used and this should remain in use for six months. Thus erythromycin, oleandomycin, chloramphenicol, and kanamycin had been successively used. Since doing this there had been no build-up of antibiotic resistance, which was a very pleasing result. But it had also been hoped that by using tetracycline as little as possible the percentage of bacteria resistant to this antibiotic would be reduced. This hope had in fact been realized, although the sub-committee was still not ready to recommend the reintroduction of tetracycline.

Another finding of great interest had been that the routine use of hexachlorophene had produced a great fall in the rate of clinical infection, and also in the nasal carrier rate. In premature babies, however, as the incidence of resistant staphylococci had fallen that of *Bacterium coli* had increased. The reason for that was not known.

DR. B. NEAL said that a recent American report had suggested that the humidifying equipment in use in premature nurseries provided favourable conditions for the multiplication of *Bacterium coli*.

MR. P. JONES said that one could sometimes trace the spread of infection in a home following the entry into it of a new-born infant.

DR. J. COLEBATCH made a plea for the greater use of sulphonamides. "Gantrisin" had been incriminated as increasing jaundice in new-borns, but other sulphonamides had not been.

Congenital Malformations and Maternal Diet.

DR. D. PITT said that nutritional deficiency had been shown by many workers to be teratogenic to the animal embryo. Vitamins of the B group especially appeared to be essential for normal organogenesis. That was consistent with the known general functions of the B vitamins, which, acting as intracellular enzymes, promoted rapid cell growth, for example, in the adult hæmopoietic and gastro-intestinal systems.

An attempt had been made to detect deficiencies in the diets of 99 mothers who had borne malformed children. When comparing their estimated diets with those of a matched control series, statistically significant differences had been found in several groups of malformation especially cleft lip, congenital heart disease, and talipes. No differences had been found in mongolism and spina bifida. The most usual differences had been in the intake of protein, calories, and B group vitamins (thiamin, riboflavin and niacin). On comparing the findings with hereditary estimates deduced from twin studies, it appeared that the worst maternal diets were found in association with those conditions in which genetic factors seemed least important.

MR. A. M. CLARKE asked whether congenital anomalies were more frequent in times of famine.

Dr. Pitt replied that he had no data on this.

DR. McKAY said that in New Guinea the diet was low in protein but the incidence of congenital anomalies did not seem to be higher than elsewhere.

DR. H. WILLIAMS asked how accurate was the assessment of the maternal food intake, and what had been the malformation rate in Europe during the war.

Dr. Pitt replied that the assessment of maternal food intake in the study had been inexact but that no better method had been able to be devised. In Rotterdam where starvation conditions had existed during the war, the abortion rate had increased; the weights of babies born during this time had been less than average but there had been no known increase in the malformation rate.

DR. BETTY WILMOT asked what had been the thiamin-carbohydrate ratio in those mothers with a low thiamin intake, as this ratio was important.

Dr. Pitt replied that the ratio had been calculated but he thought that the absolute values gave more helpful information.

MR. D. SCHLICHT stressed the importance of intrauterine moulding in the production of talipes.

A MEETING of the Pædiatric Society of Victoria was held on November 18, 1959, at the Royal Children's Hospital, Melbourne.

Galactosæmia.

DR. H. WEITENHALL said that in December five years previously a baby boy had been born, and his young mother, who had felt particularly well during her pregnancy, had been thrilled to be able to take him home for Christmas. One month later he had been admitted to the Royal Children's Hospital. He was his mother's first baby, and she had hardly been aware there was anything the matter with him; he had been very good, but had not gained weight

and that had seemed wrong. She had breast-fed him four-hourly, and he had always been contented and slept well—perhaps too much; but he then weighed only 7 lb. 2 oz., and his birth weight had been 7 lb. 9·5 oz. On examination in hospital, the baby had been noted to be thin and pale and slightly icteric. The liver had been palpable four fingers' breadth below the right costal margin, and the diagnosis of neonatal hepatitis or atresia of the bile ducts might easily have been made, had not testing of the urine revealed a reducing substance. This had been the pointer to galactosæmia, which diagnosis had been confirmed by further investigation. Urine chromatography showed galactose to be present, associated with abnormal aminoaciduria; the blood galactose level was 216 mg. per 100 ml. and the blood glucose level 90 mg. per 100 ml. Dr. R. G. Orr reported that some slight opacities were present in both lenses, and the nucleus had a well-defined border. The serum bilirubin content was 4 units, the thymol turbidity was 1 unit, the serum protein level was 5·4 grammes per 100 ml. (albumin 3·4 grammes, globulin 2·0 grammes), the hæmoglobin value was 78% and the total leucocyte count was 16,700 per cubic millimetre, the differential count being normal. The baby's blood was Group O, Rh-positive, and the Coombs test produced a negative result.

Treatment had been to withdraw milk completely from the diet, and he was given a soya-bean feeding mixture with added sugar, cod-liver oil, calcium and vitamins. He was fortunate to have a devoted and intelligent mother, and he had been maintained on a milk-free diet ever since. His mother had shown considerable ingenuity in devising recipes for him, and his diet was varied and interesting. Butter for cooking purposes had been allowed for the first time a year previously; but he still had "Nutalex" made from vegetable oils to spread on his milk-free bread. He had no chocolates or ice creams, but was allowed barley sugar and "icy poles". The boy himself had been most cooperative, and if offered anything by other children or strangers he always asked his mother first if he might have it. Calcium gluconate, 40 grains per day, and a multi-vitamin preparation were the only additions to his diet. His mother said that he had not been particularly difficult to manage, her worst time having been when he had had a salmonella enteritis, which had taken months to clear, and for which he had had to be readmitted to hospital in June, 1956.

Dr. Wettenhall said that the family history was unusual. The baby's father suffered from Huntington's chorea. So far as Dr. Wettenhall knew, that association had not been noted previously; but he wondered if it was significant. There were no siblings, but both parents had been tested. They had been shown not to have evidence of galactosæmia in any form. The patient now appeared to be a completely normal boy weighing 17 kg. and being 106 cm. tall—figures well within the average range for his age. It was reasonable to be hopeful about the prognosis from the point of view of galactosæmia; but the shadow of Huntington's chorea remained.

Correspondence.

GENERAL PHARMACEUTICAL BENEFITS.

SIR: The Federal Council has appointed a special subcommittee to consider alternative schemes which would be more acceptable to the profession, so that it can negotiate with the Government for a new and better pharmaceutical benefits service to the community. It has also been suggested by the Commonwealth Department of Health that it is prepared to accept amendments to the regulations under the present scheme, which would have the effect of removing many of the pin-pricking details to which the profession is obliged to adhere in its prescribing. Details of this relaxation of regulations are not yet official, but there is a danger that both the profession in general and the Federal Council subcommittee in particular may be inclined to accept such changes as a removal of most of our objections to the present scheme.

This is a danger to which the profession must remain alert. In my opinion, the present Pharmaceutical Benefits Scheme is wrong in principle and no amount of easing of irksome regulations within the present framework will make it a good scheme. Moreover, we will still be subject to reintroduction of unnecessary bureaucratic interference with methods of prescribing at any time in the future. The big mistake in the present scheme is the amalgamation of two previously distinct schemes, which cannot satisfactorily be mixed together; on the one hand is a very extensive list of drugs and compounded medicines, ointments, etc., many of which are quite inexpensive, which are included as benefits only because it is felt that they should be available free to pensioners; whereas on the other hand is a much smaller list of so-called life-saving and disease-preventing drugs which were hitherto free to all sections of the community, subject to certain restraints on prescribing. All drugs in both lists are now subject to 5s. charge for each prescription except in the case of pensioners.

It is the imposition of the 5s. charge to the extended list previously available only to pensioners which makes the new scheme unwieldy, unnecessary and in many instances simply stupid. It is ridiculous that drugs which used to be sold at 5s. or even less be included in such a scheme at all. It is unnecessary except for pensioners that prescriptions worth, say, 6s. to 8s. 6d. be included as a benefit. It is stupid that many drugs would be cheaper if bought privately by the hundred than they are when prescribed as a benefit in maximal allowable quantities, such as 25 tablets.

The profession should insist on separation of the two incompatible partners in this shotgun marriage. The simplest way of achieving this would be to return to the previous system of two separate lists of benefits, the only change being that drugs on the limited list available to the general community would now be subject to a 5s. charge, except to pensioners. I believe, however, that such a solution would not be satisfactory to the Government or to the profession. The profession should seize this opportunity of ridding itself of unwarranted intrusion into its methods of prescribing, which was present even in the old scheme.

Let us consider first pensioner pharmaceutical benefits, and let us agree that it is desirable that pensioners should obtain all necessary drugs and medicines in reasonable quantities without charge. We should take the stand that all prescriptions for pensioners should rank for benefit, with the exception of new and non-official remedies not yet accepted by the Pharmaceutical Benefits Advisory Committee. That being so, we should insist that we be completely untrammelled as regards quantity prescribed and number of repeats; in fact, we should insist that this particular Pharmaceutical Benefits Scheme is none of our business and would be quite easily administered between Government and chemist without regulations which concern doctors. Even the proof of pensioner entitlement can easily be done at the chemist level. We should not be concerned whether the Government or chemists like this proposal or not—it is a matter of vital principle, and the only real way to retain prescribing freedom in an all-embracing benefits scheme.

As regards the more limited list of drugs for the general community, we should insist firstly that it be severely pruned. This list should contain only genuine life-saving and disease-preventing drugs which are also costly. Restrictions as to allowable quantities and repeats should be liberalized so that the amount needed for an average patient for a reasonable period be allowable. Restriction according to the nature of the disease should not be accepted except in the case of potentially dangerous drugs, such as the cortisone group. To tell doctors that a particular antibiotic may be prescribed as a benefit for particular diseases is not only wrong in principle, but is also impossible to enforce—the less honest practitioner takes no notice of such restriction. Granted the above alterations to a limited list of life-saving and disease-preventing drugs, we should agree to help to administer such a scheme for the Government. It is no concern of ours that the patient has to pay 5s. per prescription.

If, however, the Government insists that it cannot now go back to a limited list for the general public after having allowed the present extended list of benefits, the profession should take the same stand as I have advocated in the case of pensioners—viz., that any extended list should include all approved drugs, and that there is no good reason why such a list cannot be administered at the Government-pharmacist level on a doctor's ordinary prescription. There should be no restrictions on the doctor's prescribing habits or requirements as regards quantity. Even under such a scheme, it might be necessary to have a very small list of drugs which could only be obtained when the doctor indicated the nature of the disease.

The proposals outlined above may seem somewhat drastic; but I believe we must insist on an absolute minimum of restrictions on prescribing freedom, and should take the firm stand that basically the provision of pharmaceutical

benefits is a transaction between a paternal Government and the community, with the chemist as the agent of the Government, and it should involve the doctor only in the barest minimum of cooperation.

I hope that the profession, acting through its local associations and special groups, will continue to exert pressure on its Councils in all States, because only then can Federal Council carry the fight to the Government with a sufficiently strong voice. If I may be allowed to summarize my proposals, they are as follows: (i) that the present extended Pharmaceutical Benefits Scheme is wrong in principle and should still be unacceptable even if prescribing pinpricks are removed; (ii) that any scheme of an all-embracing nature should be administered solely at Government-pharmacist level, whether it be for pensioners or general public; (iii) that the only type of scheme which the profession is willing to administer on the Government's behalf is one which is strictly limited to life-saving and disease-preventing drugs which are also costly in nature; and (iv) that even in such a limited scheme we retain the right to prescribe the quantity which we consider necessary for a reasonable period of treatment, and that we object to dictation as to the nature of the disease for which a particular drug is prescribed except in the case of potentially dangerous drugs such as cortisone.

One thing seems certain—the Government will not alter the general pharmaceutical benefits section of the Act unless the profession digs in. Let us unite in fighting for professional freedom.

175 Macquarie Street,
Sydney.
August 25, 1960.

Yours, etc.,
E. S. STUCKEY.

GEOMEDICAL AND GENETICS CONFERENCE.

SIR: I have been asked by Dr. Leonard T. Kurland, Chairman, Commission of Biometry and Genetics, Bethesda, U.S.A., to bring to the notice of your readers a Geomedical and Genetics Conference to be held in Rome on September 13, 1961.

This conference will meet in conjunction with the International Congress of Neurology and the International Congress of Genetics. The subjects to be discussed will be related to statistical methods and analysis of biological data, and the epidemiology and genetics of diseases of the nervous system.

Selby House,
87 Wickham Terrace,
Brisbane.
August 23, 1960.

Yours, etc.,
JOHN M. SUTHERLAND.

STUDY IN THE EFFICIENCY OF VACCINATION AGAINST INFLUENZA IN TWO EMPLOYMENT GROUPS IN MELBOURNE IN 1959.

SIR: The article by Dr. A. E. Duxbury and Dr. T. E. B. Keen, "Study in the Efficiency of Vaccination against Influenza . . ." (MED. J. AUST., August 6, 1960, page 206) calls for some comment. The details of the appropriate design of field trials of vaccines in general and of influenza vaccines in particular have now been well established. The trial reported seems deficient in many respects.

Firstly, it is as a general rule, undesirable to carry out mass vaccinations in the presence of an outbreak of acute infections, unless the danger threatened by the malady against which the vaccine is directed is so great that it outweighs the seriousness of the increased severity of the reactions to be expected in such circumstances. Outbreaks of epidemics due to influenza viruses Types A or B in May in Victoria, and proven by laboratory tests, occurred in 1942 (Burnet, Beveridge, Bull and Clark[1]), in 1946 (Anderson and Burnet[2]), in 1948 (Anderson[3]), in 1950 (Anderson, Donnelley, French and Kalra[4]), in 1954 (McLean, White and Stevenson[5]), and in 1957, when the Asian virus appeared at Bonegilla.

Secondly, the diagnostic criteria employed leave much to be desired, relying as they do on answers to a *questionnaire* completed from memory some several months after the events and consisting of an undisclosed proportion of self diagnoses and hearsay reports of physicians, as to the nature of the illnesses concerned. Out of these dubious data, the term "influenza" creeps into the argument without any qualification. The word "influenza" in a report of this kind should be used *sensu stricto*—that is, when there is laboratory proof of the presence of a *Myxovirus influenzæ* among a sufficiently representative proportion of the population under survey. Because it is amply documented in the technical literature that the mode of attack of influenza viruses in an area can be markedly saltatory in nature, and also when present in a closed community, may be responsible for a widely varying proportion of the cases of acute respiratory infections popularly described as "flu" occurring therein (the rest being due to any one or other of the 30 or more agents known to be responsible for such conditions), it is unwise to extrapolate laboratory findings from other sources to the population under consideration.

Thirdly, regarding the groups studied, there is no indication of the distribution of the numbers of outdoor and indoor workers or of the various age groups, both of which factors markedly influence the incidence of influenza. Further, the numerous pitfalls possible in investigations of this nature created by the use as controls of non-volunteers have been repeatedly pointed out. One of these, not so well known, may be mentioned. Some years ago, when engaged in a similar survey, the writer, while examining the incidence of absenteeism due to illness among a population of some 3500, enjoying certain rights of sick leave, observed marked peaks contemporaneous with popular mid-week sporting events, at times when no known epidemics were circulating. Whether such a phenomenon was present in the 1959 period, and if so, how extensively it was operative in one or both groups, is not deducible from the material supplied. Both populations A and B were employed within the city of Melbourne and were presumably drawn at random from the various suburbs, and had doubtless a large proportion of their members in daily contact with the general public. Therefore the disparity noted in July should have given cause for some reflection as to whether the primary data were defective. Here the absence of laboratory control is felt. Maybe Group A, being as they were servants of the tax-gatherer, were in answer to the prayers of the sinners visited by a plague and pestilence. This feeling of uneasiness as to the validity of the primary data is supplemented by the extraordinarily high protection rate (89%) claimed against a hypothetical Sendai virus invasion in face of the small dose of antigen employed (50 CCA) against the usual 60% to 80% for *M. influenzæ* A and B, after 500 CCA subcutaneously which, in the case of these more adequately documented infections, has been considered to be the desirable dosage.

It has been repeatedly shown in the past, for *M. influenzæ* A and B, that vaccination with sufficient dose of antigen, prepared from a closely related virus strain in the correct phase, after two to four weeks, for an at present uncertain period of time, will give up to 80% protection against infection by a closely related variant virus. This is a very satisfactory position to be in, against two well-known causes of severe acute respiratory infection. Any future investigations of the efficacy of influenza vaccines should be so designed that, having due regard for experience gained in the past, the maximum amount of valid information can be obtained therefrom. Such, in the opinion of the writer, would not appear to have been so, in the article criticized by him.

The subject is of some considerable national economic importance, and the central problem, now rendered somewhat less urgent by the availability of antibiotics, would appear to be how to meet another major antigenic shift of the strains of the influenza viruses against which the vaccine is prepared. The hypothesis of a closed antigenic cycle will not be proven or disproven until the next major change appears. In the event of the latter happening, we would be in the same position as in 1946 and 1957, unless there has been developed in the meantime some effective form of chemotherapy against the influenza virus, or a new type of vaccine which it is technically possible to prepare and distribute more quickly than is at present possible.

For the present, a very great deal can be done to control the "ravages" of "influenza" by public health authorities not lending their support to hysterical panic-raising campaigns in the various forms of communicating information to the public. They only serve to cause unwarranted fears among the people and to disrupt medical practice. Chapter and verse for this view can be taken from the *Australian Demographic Review*, Commonwealth Bureau of Census and

[1] MED. J. AUST., 1942, 2: 371.
[2] *Aust. J. exp. Biol. med. Sci.*, 1947, 25: 235.
[3] *Aust. J. Sci.*, 1948, 11: 59.
[4] MED. J. AUST., 1953, 2: 44.
[5] MED. J. AUST., 1955, 1: 257.

Statistics, Numbers 81 and 82, 1956 and 1957, which indicate that, between the year 1952 and the year of the advent of the Asian strain of influenza, in the crude mortality rate for deaths from all causes in Australia, the figure for 1957 was the lowest. Further, in the years 1956 and 1957, during the epidemic months June to October inclusive, the total monthly deaths from all causes in 1957 exceeded those for 1956 only in September (7930:7997). It is not desired to minimize the seriousness of the morbidity due to influenza, but merely to point out that there is no cause for undue alarm while efforts are being continued in various quarters to find, as no doubt will be found, the final solution to the problem.

98 Nicholson Street,
Fitzroy,
Victoria.
August 21, 1960.

Yours, etc.,
L. C. ROWAN.

BRUSH UP YOUR MEDICINE: OTITIS MEDIA AND OTITIS EXTERNA.

SIR: I would like to support Dr. Bulteau's letter published by you on August 13, 1960, on the treatment of otitis media and externa.

The treatment of otitis media by the general practitioner must be effective in view of the few cases and complications the specialist sees nowadays, while the treatment of otitis externa by simple drugs suitably applied is very effective.

In a paper entitled "Methods of Treating Otorrhœa and Some Common Diseases of the External Ear" I read at the North Queensland Medical Conference at Mackay in 1958, I said:

> The commonest errors in treating the running ear are:
>
> 1. Failure to treat on an ætiological basis.
> 2. Failure to do an effective ear toilet.
> 3. Failure to treat on a basis of physiological pharmacology.
> 4. Using auristillas with water as a vehicle.
> 5. Using pigments such as gentian violet. These are not antiseptic in ears, soil the pillow, and obscure the view. To some this may seem their main attraction.
> 6. Using glycerine of ichthyol. Ichthyol is pharmacologically inert, and its unpleasant colour offends the patients. It is in the same category as vipers' tongues, newts' livers and the caul.
> 7. Depending on antibiotics locally and parenterally particularly:
>
> (*a*) Using penicillin and sulpha powders in the external canal. They are ineffective against many organisms, particularly the commonest found in aural infection, *Pseudomonas pyocyanea*, and no surer way of sensitizing patients to both drugs could be devised.
>
> (*b*) Parenteral penicillin injections which are rarely effective and can also produce sensitivity. The diagnostically destitute and the lazy or overworked immediately use penicillin forgetting its narrow spectrum and the patient's possible existing sensitivity.
>
> The attitude of mind should be one of opposition to the use of antibiotics.
>
> 8. The misuse of an aural syringe. Lavage will reinfect a middle ear through a perforated membrana tympani and irritate an allergic otitis externa.

Yours, etc.,
R. PALMERSTON RUNDLE.

40 Denham Street,
Townsville,
North Queensland.
August 23, 1960.

THOUGHTS ON MEDICAL CARE.

SIR: May I offer the following thoughts on medical care for suggestions and comment?

1. All hospital treatment would be free. The Commonwealth would support all hospitals of acceptable standard, which would operate in much the same form as at present. The "public" hospitals woud be staffed by residents and registrars, with consultant staff paid on a sessional basis, and would train nurses and doctors, do medical research and have specialized techniques—e.g., for hypothermia. The consultants would be primarily in private practice as at present, and the system would operate much as at the Royal Children's Hospital, Melbourne. The private hospitals would be available for treatment of patients by any doctor, general practitioner or consultant, as at present, except that there would be no hospital bills for the patient.

2. Doctors' fees would be paid directly to the doctor by the patient, and the latter would be reimbursed to the extent of 90% or 95% by the Commonwealth. Voluntary insurance schemes, due to human laziness, carelessness or penury, will never cover 100% of the population, and a compulsory addition to tax would be more equitable.

3. Any prescription costing more than five, or ten, shillings would be subsidized by the Commonwealth. There would be no restriction on the drugs prescribed.

31 Rosslyn Street,
East Hawthorn, E.3,
Victoria.
August 20, 1960.

Yours, etc.,
J. E. GAULT.

BRUSH UP YOUR MEDICINE: NON-MERCURIAL DIURETICS.

SIR: In your issue of August 13, "Brush Up Your Medicine —Non-Mercurial Diuretics", by Alex Johnson, we see you were good enough to give a tabloid review of our product "Cardrase".

We realize that in this type of presentation the limitation of space can cause certain restrictions on fullness of detail, and unhappily this has caused the dosage of "Cardrase" to be shown as two to four tablets (on three consecutive days each week or on alternate days). The recommended dosage for "Cardrase", apart from its conjoint use in glaucoma and epilepsy, is one to two tablets (on three consecutive days each week or on alternate days), and is usually one tablet. The administration of twice this dosage may lead to undesirable side-effects, some of them brought about by too powerful a diuretic action.

Yours, etc.,
for The Upjohn Co. (Aust.) Pty. Ltd.,
F. P. COLLINGS.

55-73 Kirby Street,
Rydalmere, N.S.W.
August 25, 1960.

A COMPARATIVE STUDY OF PREGNANCY AND LABOUR IN PRIMIPARÆ OF INCREASING AGE.

SIR: The author of this article, which appeared in the MEDICAL JOURNAL OF AUSTRALIA on August 20, 1960, is to be congratulated on presenting a detailed statistical survey of the problem as shown in the figures from the Women's Hospital, Crown Street. I cannot, however, agree with his suggestion that in his series the primipara becomes "elderly" at the age of 37 years. His age groups were (i) 15 to 18, (ii) 25 to 28, (iii) 35 to 37 and (iv) 38 and over.

Taking the figures from his own article, we find the following changes when we compare his 35 to 37 year group with the average of his two groups less than 28 years of age:

1. Cases seen before 16 weeks: (*a*) average blood pressure increased 4·5 mm. of mercury; (*b*) essential hypertension increased 9·5%.
2. Essential hypertension of the whole group increased 5%.
3. Ante-partum hæmorrhage 3% compared with 1%.
4. Spontaneous onset of labour decreased 5·5%.
5. Incidence of induction of labour increased 4·5%.
6. Incidence of prolonged labour increased 9%.
7. Duration of the second stage of labour increased in average time by 48 minutes.
8. Incidence of forceps delivery increased 26%.
9. Cæsarean section rate 5% as compared with 0% in the younger groups.
10. Postpartum hæmorrhage increased 5%.
11. Manual removal of the placenta increased 8·5%.
12. The incidence of prematurity as shown on the graph is increased.
13. The final burden on the 35-year-old is that the number of live births per 100 pregnancies falls by seven.

These 13 points are suitable in number and speak for themselves. If we are to use a term to signify an increased risk with age to the primipara, it must on this series be

applied to those older than 35. A decrease in live births of seven per 100 pregnancies is a great loss. We must remember that the women over 35 are the ones who frequently have been infertility cases and may not again become pregnant easily. Thus they may then remain childless.

It may not seem polite to refer to a 30-year-old woman as elderly, but I think that if the author had compared figures of two groups, one over and one under the age of 30, he would have found a definite statistical difference in results. If we do not wish to refer to a 30-year-old as "elderly", it is the term that is at fault, not the age at which we apply it.

In this article we are told that 2 babies died from tentorial tears due to vaginal extraction through a central placenta prævia, and that the average duration of the second stage in group (iii) was 2 hours. These statements seem surprising in a report from a modern hospital.

"Wickham House",
Wickham Terrace,
Brisbane.
August 23, 1960.

Yours, etc.,
KEITH FREE.

THE MANAGEMENT OF VARICOSE ULCERS.

SIR: I read with interest the article by Mr. W. Stern in the Journal of August 20, 1960, on the above subject, in which he stresses nothing new has been brought to light since 1931, when Mr. Dickson Wright advocated compression bandaging of the legs for the control of ulceration.

The article confirms the view widely held by sufferers of varicose ulcers and the great majority of the medical profession that the patient has to embark on a life of continuous daily bandaging of the legs, renewal of elastic stockings every three months and attendance at varicose vein clinics.

With the passage of years I have become adjusted to refrain from any criticism of present management of ulcers; but I could not ignore this article, which contained so much out of date knowledge and vague generalities in treatment. There is only one answer to the problem, and that is to correct the venous flow in these legs through surgery.

Quote from the article: "Local or generalized varicosities have often suffered from the enthusiastic procedures of surgeons without the ulcer being cured." In my practice I am pleased to see any patient with an ulcer associated with large incompetent saphenous veins and perforating veins, as you can assure that patient he will be cured of that ulcer in two months and be able to discard further elastic support.

Cockett recently demonstrated to me the amount of blood which passes from the deep veins to the superficial saphenous vein through an incompetent perforating vein. From my own observations, it is desirable one or both legs should be completed at one operation. This includes removal of both the long and short saphenous veins, all tributaries in the groin, any large tributaries of the long and short saphenous veins, communicating and perforating veins when they are diseased.

To most surgeons this is impossible under local anæsthesia. My operating table has been made comfortabe, so the patient sleeps most of the time and can cooperate when necessary. The operation takes four hours in bad cases and less in simpler cases, with the loss of 2 oz. of blood during the whole operation.

The patients are able to walk after the operation and are completely ambulant next day. The wounds heal by first intention without antibiotics, and the ulcer rapidly disappears. Bandages are discarded in two months permanently in 95% of cases; 5% have such gross pathology present from long-standing infection and ulceration that bandaging is the only form of relief.

From 985 cases in ten years' operating, 261 cases were put on the table with an ulcer present on the leg, 120 had previous operations elsewhere, 20% were between 60 and 70 years. Patients resume work in 14 days and heavy work in 21 days.

One patient died from a pulmonary embolism on the sixteenth day following stripping of the short saphenous vein, which in my opinion is not safe.

All bleeding points are tied throughout the operation, which prevents clots, emboli and pain. No blood transfusions

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 13, 1960.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	3(2)	3(2)	..	1(1)	..	..	..	7
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	1	..	..	..	..	..	7	..	8
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3(3)	25(22)	6(3)	..	1(1)	1(1)	3	..	39
Diphtheria	1	..	..	..	..	..	..	..	1
Dysentery (Bacillary)	..	1(1)	..	..	6(2)	..	2	..	9
Encephalitis	..	..	..	2	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	1	..	..	1
Infective Hepatitis	87(29)	44(20)	14(1)	20(14)	7(3)	..	..	1	173
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	4	..	4
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	1	1(1)	..	..	..	..	2
Meningococcal Infection	..	..	..	..	..	..	..	..	..
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	..	..	..
Puerperal Fever	3	..	..	..	..	..	..	..	3
Rubella	..	7(4)	..	..	..	..	..	..	7
Salmonella Infection	..	..	..	6(1)	..	..	..	..	6
Scarlet Fever	25(17)	15(11)	4(3)	..	..	..	..	..	44
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	..	..	..	..	..	..	1
Trachoma	..	..	..	..	1(1)	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	41(24)	18(16)	14(3)	4(3)	3	1(1)	..	..	81
Typhoid Fever	1	..	..	..	..	..	1	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

were given. Patients do not receive sclerosing injections subsequently which does more harm than good in the long run.

Having used the standard stripper in some cases and watched it used in Britain, one is impressed by the number of unsatisfactory jobs where most of the vein is left behind.

This type of work should be done by trained surgeons who are willing to devote enough time to the complete operation, otherwise there will be no progress in this field of surgery.

Yours, etc.,
J. N. R. STEPHEN, M.B., Ch.B.

A.N.Z. Chambers,
229 Hunter Street,
Newcastle, N.S.W.
August 25, 1960.

Australian Medical Board Proceedings.

MEDICAL BOARD OF VICTORIA.

THE following statement is published at the request of the Medical Board of Victoria.

The Medical Board of Victoria recently sustained charges of infamous conduct in a professional respect against two Melbourne doctors for having improperly signed yellow fever vaccination certificates contained in World Health Organization booklets.

As yellow fever vaccine can only be administered by Commonwealth Medical Officers the irregularities were immediately noted when the booklets were presented for endorsement at the Commonwealth Department of Health office in Melbourne. The Commonwealth Government, as a signatory to the International Sanitary Convention, is required to accept responsibility for the correctness of particulars entered in the booklets.

On one charge Dr. R. P. Strang, of 14 Dunraven Avenue, Toorak, was reprimanded, and two charges against Dr. Tom Sieber, of 87 Chatsworth Road, Prahran, resulted in the suspension of his registration for three months.

Corrigendum.

REVIEW: SYNOPSIS OF EAR, NOSE AND THROAT DISEASES.

IN the review of "Synopsis of Ear, Nose and Throat Diseases", by Robert E. Ryan on page 384 of our issue of September 3, 1960, the last sentence of the third paragraph should read as follows: "The use of Boyle-Davis gag and the extended head position are not mentioned by these authors, who favour intubation of the trachea in children with the patient lying flat."

Nominations and Elections.

THE following has applied for election as a member of the New South Wales Branch of the British Medical Association:

Hidi, Madelaine, M.D., 1953 (Univ. Budapest), licensed under Section 21C (4) of the *Medical Practitioners Act*, 1938-1958, 41 Burwood Road, Concord.

The following have applied for election as members of the South Australian Branch of the British Medical Association:

Barclay, Evelyn Frances, M.B., Ch.B., 1957, D.C.H., Adelaide Children's Hospital, North Adelaide, S.A.

Rozenbilds, Jeff Girts, M.B., B.S., 1959 (Univ. Adelaide), Royal Adelaide Hospital, North Terrace, Adelaide.

Baillie, James Gibson, L.R.C.P.I., L.R.C.S.I., 1958, Crozier Street, Victor Harbour.

Coulter, Carol Gladys Roberta, L.R.C.P.I., L.R.C.S.I., 1957, Crozier Street, Victor Harbour.

The following have been elected as members of the South Australian Branch of the British Medical Association:

Filipic, Marijan, M.B., B.S., 1959 (Univ. Adelaide); Ludlow, John, M.B., B.S., 1959 (Univ. Adelaide).

Diary for the Month.

SEPTEMBER 19.—Victorian Branch, B.M.A.: Finance Sub-Committee.
SEPTEMBER 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPTEMBER 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPTEMBER 22.—Victorian Branch, B.M.A.: Executive Meeting of Branch Council.
SEPTEMBER 23.—Queensland Branch, B.M.A.: Council Meeting.
SEPTEMBER 24.—Victorian Branch, B.M.A.: Clinical Meeting (Children's Hospital).
SEPTEMBER 27.—New South Wales Branch, B.M.A.: Hospitals Committee.
SEPTEMBER 27.—Tasmanian Branch, B.M.A.: Southern Subdivision.
SEPTEMBER 28.—Victorian Branch, B.M.A.: Branch Council.
SEPTEMBER 29.—Tasmanian Branch, B.M.A.: Northern Subdivision.
SEPTEMBER 29.—South Australian Branch, B.M.A.: Scientific Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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